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Comments Submitted on Behalf of Universal Oil Products, Inc. ("UOP") Relating to the Torch Lake Superfund Site

Preliminary Statement

After four years of study, the United States Environmental Protection Agency ("EPA") has produced a large body of information demonstrating that the Torch Lake Superfund site in Michigan's Upper Peninsula is a healthy environmental resource that poses no significant risk to the area's residents, visitors or wildlife. Rather than welcoming this result, EPA has instead ignored virtually all the data in the record and mischaracterized the rest in an apparent attempt to justify a multimillion dollar "remedy"

that is neither necessary, nor wanted by the affected community, nor lawful under the CERCLA.[1/]

As required by CERCLA, EPA has solicited comments from the public on its Proposed Plan for remediation. This memorandum, together with comments by Geraghty & Miller, a nationally recognized firm of environmental scientists and engineers, is submitted on behalf of UOP Inc. to bring to EPA's attention the uncontroverted data in the Administrative Record demonstrating that no remedial action is appropriate with respect to the Torch Lake site.

As is more fully set forth below, adoption of the Proposed Plan would be unlawful under CERCLA because EPA's soil and vegetation remedy does not advance the statute's goals or comply with EPA's own regulations implementing the statute.[2/] As demonstrated in the Administrative Record,

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1/ The Comprehensive Environmental Response, Compensation, and Liability Act, 42 U.S.C § 9601 et seq. (hereinafter "CERCLA" or the "Superfund Law").

2/ Specifically, as demonstrated herein and in more detail in the Geraghty & Miller comments, when compared to the no action alternative, the Proposed Plan fails to advance CERCLA's goals to any meaningful degree as measured by the National Contingency Plan's threshold and primary balancing criteria: (1) it fails to provide overall protection of human health or the environment to a significant degree beyond that provided by the no action alternative; (2) it fails to assure compliance with any applicable or relevant and

(continued...)

the no action alternative satisfies the National Contingency Plan ("NCP") threshold criteria, and based on the NCP balancing criteria, it is more appropriate than the proposed remedy for Torch Lake. The no action alternative is protective of human health and the environment, attains all applicable or relevant and appropriate requirements ("ARARs") and, obviously, is more cost effective than the Proposed Plan. In situations such as this one, the NCP requires EPA to base its selection of a remedial action on cost effectiveness: "Each remedial action shall be cost effective, provided that it first satisfies the threshold criteria [of overall protection of human health and the environment and compliance with ARARs]. . . . A remedy shall be cost effective if its costs are proportional to its overall effectiveness." 40 C.F.R. § 300.430(f)(1)(ii)(D).

[2/] (...continued)
appropriate requirements beyond that assured by the no action alternative; (3) it fails to advance long-term effectiveness and performance goals to a significant degree beyond that advanced by the no action alternative; (4) it fails to reduce toxicity, mobility or volume through treatment; (5) it is less protective than the no action alternative of short-term effectiveness goals; (6) it will be considerably more difficult and expensive to implement than the no action alternative; and (7) it is considerably less cost effective than the no action alternative. 40 C.F.R. § 300.430(f)(1)(i).

In rejecting the no action alternative, EPA ignored uncontested data in the record demonstrating that the site does not present any unacceptable risks to human health, that wildlife and the environment show virtually no adverse affects from tailings and slag, and that EPA's remedy does not, in any way, address the agency's claims of harm to health or the environment. Moreover, EPA fails adequately both to explain how implementing the Proposed Plan will not cause more environmental degradation than it is intended to cure and to examine the Proposed Plan's long-term effectiveness.

The Geraghty & Miller comments demonstrate that EPA's justifications for its proposed soil cover and vegetation remedy consist of either outright misstatements of fact ("The reproduction of Yellow Perch has been hampered by the continuous release of contaminants into Torch Lake." -- There is no support for this in the record), misleading mischaracterizations of the record ("Bald eagle nests have been identified that may be threatened." -- EPA's own studies show this is false (USFWS 1991 - A.R. Doc. No. 191)), or inherently contradictory double speak ("[U]nder a no action alternative . . . RAOs [Remedial Action Objectives] . . .

will not be met." -- This statement is false, as demonstrated by EPA's statement on the same page that "because human health risks for OUI and OUIII are generally within the acceptable range . . . no action is a feasible alternative").

This country faces very real environmental problems, but Torch Lake is not one of them. In an era of limited financial resources, it should be inconceivable that a federal agency would allocate vast sums to an unnecessary action on the basis of such a record. We urge EPA to discharge its responsibilities under CERCLA and address in detail each of the Geraghty & Miller comments and those expressed herein. Based on a fair and reasoned examination of the Administrative Record, EPA must conclude that no action is the only alternative that lawfully can be selected for Torch Lake.

I. Background

A. Site History

Michigan's Upper Peninsula is one of the few locations in the world where copper is found in virtually pure form. As a result, for over 100 years the area, known as the Copper Range, was the most productive copper producing area in the United States. Mining and smelting operations

began in the Copper Range before the American Civil War, grew dramatically until the 1930s, and then gradually diminished and ended in the 1960s.

Some by-products of the recovery of 5 million tons of copper are large amounts of ore with low metal content ("poor rock"), sands from the crushing of copper containing ore ("stampsands"), and slag (molten rock) from copper smelting operations. In the Upper Peninsula, there are large areas of poor rock, stampsands and slag.

Stampsands are generally found in water bodies (e.g., Lake Superior, Torch Lake, Portage Lake and Dollar Bay) because they were slurried for disposal pursuant to federal permits. See, e.g., Appendix to Comments Submitted on Behalf of Universal Oil Products, Inc. by Paul, Weiss, Rifkind, Wharton & Garrison (hereinafter "Appendix") at Exhibit K. By its very nature, the area's soils are rich in copper and other metals, and the distinction between stampsands and other sands is often imperceptible.

B. EPA's Listing of the Site on the National Priorities List

In the early 1970s, students at a local university conducted a study of growths in fish netted out of Torch Lake. The study was alarming. It suggested that sauger and

walleye taken from the lake possibly had cancerous growths. In 1983, as a result of this study, the Michigan Department of Health issued an advisory against consumption of fish caught in the lake. Thereafter, the Michigan Department of Natural Resources ("MDNR") asked EPA to list Torch Lake as an "area of concern" in the Great Lakes Program and to consider listing the lake and surrounding area on CERCLA's National Priorities List ("NPL").

EPA nominated the Torch Lake area as an NPL site in 1984. The site was listed on the NPL in 1986 primarily as a result of the theoretical possibility that the surrounding communities could be exposed to contaminated groundwater. There was, however, no evidence of contamination of any water supply in the area; nor has any such evidence emerged.

C. MDNR's Evaluation of Torch Lake Fish

Between 1980 and 1989, MDNR systematically evaluated the fish in Torch Lake. MDNR's studies clearly demonstrate that Torch Lake is a healthy and safe environment with a normal fish population. For example, MDNR's evaluation of the fish in Torch Lake found the following:

- *Not one of 455 Torch Lake fish which were caught in 1988 had any tumors (BLACK 1987 and TVA 1987 - A.R. Doc. No. 67);*

- *Torch Lake fish are among the least contaminated of all the fish from lakes studied in Michigan (MDNR 1990 - A.R. Doc. No. 118);*

- *A widely publicized study in the 1980's stating that fish tumors were increasing was without merit (MDNR 1987 - A.R. Doc. No. 56; MDNR 1989 - A.R. Doc. No. 67), and the mid-1970's study finding cancerous growths in fish was, in all likelihood, incorrect as well;*

- *No significant concentrations of cancer causing substances have been found in Torch Lake (MDNR 1989 - A.R. Doc. No. 108);*

- *Torch Lake, based on MDNR and EPA studies, is safe for swimming and has had no significant adverse affect on fish (MTU 1984 - A.R. Doc. No. 41; MDPH 1984 and WUPDHD 1984 - A.R. Doc. No. 40); and*

- *Studies show that stampsands, in fact, have released almost no contaminants into Torch Lake (USDI 1991a - A.R. Doc. No. 172; USDI 1991b - A.R. Doc. No. 194; Rose et al. 1986 - A.R. Doc. No. 50).*

D. EPA's Study of the Site

EPA began preparing the Torch Lake site remedial investigation and feasibility study ("RI/FS") in 1988. The site was divided into three "Operable Units." Operable Units I and III are, respectively, Torch Lake's western shore and twelve distant tailings and slag locations in the Upper Peninsula. Operable Unit II consists of the waters of Torch Lake, tailings and sediment at the bottom of the lake and the site's groundwater. The final RI/FS report on Operable Unit II is scheduled for completion in 1993.

Like MDNR's evaluation of the Torch Lake fish advisory, the RI/FS for Operable Units I and III provides a virtual clean bill of health for Torch Lake. For example, the RI/FS contains the following conclusions:

- *Even using EPA's very conservative method of assessing cancer risks, no significant cancer risk exists (G&M 1992a; G&M 1992b). Based on overly conservative assumptions about ingesting slag and stampsands, EPA concluded that the increased risk of cancer was less than that caused by smoking a few cigarettes over a lifetime (Donahue 1992 - A.R. Doc. No. 198). In fact, EPA acknowledges that these risks are not sufficient to justify any action;*

- *Torch Lake area residents do not display any adverse health effects as a result of poor rock, slag or stampsands or water which has come into contact with these substances (ATSDR 1989 - A.R. Doc. No. 73);*

- *Stampsands have no significant potential for leaching copper or other metals into groundwater (USDI 1991a - A.R. Doc. No. 172; USDI 1991b - A.R. Doc. No. 197; Rose, et al. 1986 - A.R. Doc. No. 50). Nor are stampsands affecting Torch Lake's water quality;*

- *Torch Lake area eagles and gulls have suffered no demonstrated adverse impacts (USFWS 1991 - A.R. Doc. No. 191);*

- *Studies of fish confirm that the Torch Lake fish population is not contaminated (MDNR 1986 - A.R. Doc. No. 52; MWRC 1970 - A.R. Doc. No. 17; MDNR 1990 - A.R. Doc No. 118); and*

- *Water and wind are not significant vehicles for the transportation of stampsands or slag materials into Torch Lake (MDNR 1987 - A.R. Doc. No. 56; Rose, et al. 1986 - A.R. Doc. No. 50).*

II. EPA's Proposed Plan Violates CERCLA

One reads in vain for any clear reference in EPA's Proposed Plan for Torch Lake to these undisputed findings. Instead, EPA has ignored most of the data and mischaracterized the rest in a transparent attempt to justify a proposed remedy that requires 1,000 acres of stampsands and slag to be covered with topsoil and vegetation at an estimated cost of $7.2 million. The Administrative Record, as clarified by Geraghty & Miller's comments (G&M 1992b; G&M 1992c), demonstrates that this proposed remedy violates CERCLA.

A. There are No Unacceptable Health Risks Presented by Torch Lake; Accordingly, The Proposed Plan is Unjustifiable

EPA states that soil cover and vegetation are necessary to reduce carcinogenic and noncarinogenic risks posed by the areas to be remediated. This position is demonstrably wrong, and we specifically request EPA to explain it in light of the following comments.

1. *EPA has acknowledged in public and has stated in the FS and Proposed Plan that its hypothetical calculations of cancer risks produce numbers in EPA's acceptable range. In fact, in the Proposed Plan, EPA acknowledges that "[b]ecause the human health risk for OUI and OUIII are generally within U.S. EPA's acceptable range" the no action alternatives for the tailings and slag areas "are feasible alternatives."*

2. *Studies of Torch Lake area residents reveal no abnormal instances of carcinogenic or noncarcinogenic illness.*

3. *EPA's estimates of cancer risks, which it in fact found acceptable, were based upon unrealistic and overly conservative assumptions and were calculated in a manner that is inconsistent with EPA guidance documents in at least the following respects (G&M 1992b):*

   (a) *The fact that the slag is inedible was not considered when conclusions were drawn about the ingestion risks;*

   (b) *Period of exposure estimates are unrealistically high, and many of these estimates do not agree with EPA recommended exposure periods;*

   (c) *Soil ingestion rates are excessive as they are based on unrealistic assumptions about the exposure of workers to soil;*

   (d) *Soil and slag ingestion and inhalation rates are excessive as they fail to account for the average 149 days of annual snow cover that blankets the entire area;*

   (e) *Inhalation rates for children are inaccurate and do not follow EPA guidelines;*

   (f) *The assumption that all chromium on-site is hexavalent chromium cannot be supported in science and is overly conservative; and*

   (g) *Background concentrations of copper, arsenic, beryllium and other contaminants were not considered when assessing the risks from these contaminants. For example, soil samples from local residents' backyards, which have not been affected by the tailings or slag, show cancer risks greater than the areas slated for remediation. The failure to consider background conditions violates EPA guidance documents for risk assessments.*

4. *These unrealistic assumptions, when combined in EPA's risk calculations, improperly magnify risks by many times. The effect of this approach has been best described by EPA's former Assistant Administrator for Policy, Planning and Evaluation: "Multiplying large uncertainties associated with each factor in the estimate [of health risks] leads to cascading conservatism in decision making."* L. D. Maxim, *Problems Associated With The Use of Conservative Assumptions in Exposure and Risk Analysis, in* The Risk Assessment of Environmental and Human Health Hazards: A Textbook of Case Studies 526, 535 (Dennis J. Paustenbach ed., 1989) (See Appendix at Exhibit E). *For precisely these reasons, EPA stated in its February 26, 1992 "Guidance on Risk Characterization For Risk Managers" as follows:*

> If only limited information on the distribution of the exposure or dose factors is available, the assessor should approach estimating the high end by identifying the most sensitive parameters and using maximum or near-maximum values for one or a few of these variables, leaving others at their mean values. In doing this, the exposure assessor needs to avoid combinations of parameter values that are inconsistent, e.g., low body weight used in combination with high intake rates, and must keep in mind the ultimate objective of being within the distribution of actual expected exposures and doses, and not beyond it.

The Torch Lake Risk Assessments, in failing to conform to this guidance, follow a practice repeatedly condemned by members of the scientific community.[3/]

3/ See generally Appendix; see also Philip Abelson, "Incorporation of New Science and Risk Assessment," Science, Vol. 250, No. 4987 at p. 1497 (Dec. 14, 1990) ("Based upon its current modes of risk assessment, EPA is embarked upon programs that will cost hundreds of

(continued...)

5. *EPA's assertion that copper in stampsands and slag produces a non-carcinogenic "subchronic hazard index of more than 1.0 for children living near the slag pile beach in Hubbel" is simply wrong. Putting aside that ingestion of slag is inconceivable and that, as described above, EPA's assumptions concerning slag ingestion rates are overly conservative, the RAs simply ignore the following critical information:*

   (a) *Both the OUI and OUIII Risk Assessments state that the hazard index is not a valid indicator of non-carcinogenic risk significance at the site (OUI RA at 5-5; OUIII RA at 5-16); and*

   (b) *EPA improperly calculated in the RA a "reference dose" (RFD) for estimating toxic effects of .037 mg/kg of body weight per day. However, in the same paragraph, EPA concluded that copper, which is an essential nutrient, has a "relatively low oral toxicity to humans and intakes of up to .5 mg/kg/day (35 mg/day for an adult) are not expected to cause adverse effects (NAS 1989)" (OUI RA at 4-14). In fact, the estimated daily intake of copper on which EPA based its assertion of a hazard*

3/ (...continued)
billions of dollars, but will have little impact on human health") (Appendix at Exhibit A); C. Travis and C. Doty, "Superfund: A Program Without Priorities," printed in Environmental Science Technology, Vol. 22, No. 11 at 1333 (1989) ("[W]e found that before remediation, 70% of all Superfund sites had risk levels in . . . the same range that EPA targets as acceptable after remediation. Although estimates of future risks were often high, these estimates were based upon hypothetical exposure scenarios. . . . [G]iven the limited resources of Superfund, the immediate focus should be on identification of sites where risk is real and current") (Appendix at Exhibit D); Peter Passell, "Experts Question Staggering Costs of Toxic Cleanups," The New York Times, at p. A1 (Sept. 1, 1991) (Appendix at Exhibit F).

> *index transgression is significantly less than the recommended safe and adequate range of dietary copper intake for adults (i.e., 1.5 to 3 mg/day).* Subcommittee on the Tenth Edition on the RDAs, National Research Council, Recommended Dietary Allowances (10th ed. 1989) (Appendix at Exhibit I).

B. Torch Lake is a Healthy and Productive Environment; Accordingly, the Proposed Plan is Unjustifiable

The Proposed Plan and the FS also assert that the soil cover and vegetation remedy is necessary to protect the environment based upon the following statements: "Rain water or winds carry tailings into the surface water or sediments;" "Natural plant communities have been unable to develop because of the chemical and physical characteristics of the tailings;" "Sediment contamination has had an adverse affect on the lake bottom ecosystem;" "[Sediment contamination] may be affecting fish reproduction and population;" "The tailings piles have destroyed natural habitats, such as wetlands . . . which in turn has resulted in the loss of migratory and residential animal populations;" "[A]rsenic from OUI tailings may leach into the groundwater;" "[T]he tailings over the water's edge continue to degrade the environment and are a continuing source of contamination to water bodies;" "Bald eagles may be adversely affected by direct and indirect exposure to contaminated media at the

site . . .;" "The reproduction of Yellow Perch has been hampered by the continuous release of containments into Torch Lake."

These assertions are either directly contradicted by studies in the record or address the self-evident truth that the tailings and slag have physically altered the natural characteristics of the area. The latter observation can be said of every manifestation of man's presence on earth and is not a justification for remedial action under CERCLA.

The data in the record, as opposed to EPA's unfounded speculations, are described below. Again, we request that EPA explain how and why the Proposed Plan is necessary or legal in light of each of these studies.

1. *Studies in the Administrative Record show that stampsands do not have any significant potential for leaching metals, including copper and arsenic, into groundwater (USDI 1991a - A.R. Doc. No. 172; USDI 1991b - A.R. Doc. No. 191; Rose, et al. 1986 - A.R. Doc. No. 50). Nor do any studies show that stampsands and slag are affecting Torch Lake water quality.*

2. *The <u>only</u> relevant study in the record shows that there is no significant transportation of stampsands or slag into Torch Lake by either wind or rainwater (Rose, et al. 1986 - A.R. Doc. No. 50; MDNR 1986 - A.R. Doc. No. 56).*

3. *Studies of eagles and gulls show that birds in the area have suffered no adverse affects from stamp-*

sands or slag in the Torch Lake area (USFWS 1991 - A.R. Doc. No. 191).

4. *In 1988, MDNR collected 455 fish from Torch Lake. No tumors were identified in any of these specimens (MDNR 1989 - A.R. Doc. No. 108; MDNR 1990 - A.R. Doc. No. 118).*

5. *MDNR testing of contaminants in fish taken from Torch Lake shows that these fish are among the least contaminated of all fish in lakes studied in Michigan (MDNR 1990 - A.R. Doc. No. 118).*

6. *No significant concentrations of cancer causing substances have been found in Torch Lake (MDNR 1989 - A.R. Doc. No. 108).*

7. *Torch Lake, based on MDNR and EPA studies, is safe for swimming and has had no significant adverse affect on fish (MTU 1984 - A.R. Doc. No. 41; MDPH 1984 and WUPDHD 1984 - A.R. Doc. No. 40).*

8. *A widely publicized study in the 1980's stating that the number of tumors in fish is increasing is without merit (MDNR 1987 - A.R. Doc. No. 56; MDNR 1989 - A.R. Doc. No. 67). In addition, the mid-1970's study finding cancerous growths in fish is, in all likelihood, also incorrect (Black 1987 and TVA 1987 - A.R. Doc. No. 67).*

Simply no data in the record supports the Proposed Plan's conclusion that tailings and slag, unless covered by soil and vegetation, degrade the environment in any meaningful way. The only accurate statements in the FS and Proposed Plan relate to the fact that the presence of tailings and slag have altered Torch Lake's natural state. This alone cannot justify a remedy under CERCLA, and in any event, the proposed remedy does nothing to address that

circumstance. The record shows that there is almost no transportation by rain or wind of stampsands or slag into Torch Lake. Thus, even if one were to accept the conclusion that the benthic community was degraded, the soil and vegetation remedy does not address the issue. Nor will this remedy in any way affect wetlands except perhaps adversely because of the environmental disruption caused by implementing it.

We challenge EPA to articulate a single circumstance in which scientific data show that a soil and vegetation remedy is likely to ameliorate harm to wildlife. While it is true that growing grasses on tailings and slag could be seen by some as a laudable beautification project for the Upper Peninsula, it is in no way appropriate to mandate such a project under CERCLA.

Also, we challenge EPA to articulate, based upon scientific data in the record, a single way in which the proposed remedy will alleviate scientifically established carcinogenic and non-carcinogenic health risks which are unacceptable according to EPA criteria.

Finally, we challenge EPA to articulate, based upon scientific data in the record, any meaningful benefit

from its proposed soil and vegetation remedy that will not also be accomplished by no action.

III. The Inappropriateness of the Proposed Plan is Demonstrated by Other RODs Where the No Action Remedy was Selected

As noted above, EPA acknowledges that because the human health risks for OUI and OUIII are within the acceptable range the no action alternatives for both the tailings and slag areas are feasible. The Proposed Plan requires extensive remediation because EPA, presumably, perceives that such remediation significantly furthers the NCP goal of providing overall protection of human health and the environment to the degree necessary to make the remediation cost effective. The arbitrary nature of this conclusion is further demonstrated by the fact that EPA has chosen the no action alternative at many sites where the human health and environmental risks were at least as great as, if not greater than, those at Torch Lake.

We request that EPA explain why selecting the no action alternative at Torch Lake would be significantly less protective of human health or the environment than it is at each site where it has been selected, but, at least, at each of the following specific sites.

1. *Cecil Lindsey Site, Arkansas R06-86/009. This 5.2 acre site, located in the 10 year floodplain, received wastes for salvage and disposal from the early 1970's to 1980. The northern part of the site was used as a municipal dump; the site reportedly was used for the disposal of industrial wastes; and EPA enforcement files suggest the possibility of a substantial volume of waste at the site. Inorganic and volatile organic contamination in on-site soil exceeds background levels; on-site groundwater consistently exceeds background levels for inorganics; and off-site surface water and sediment samples contain some of the same inorganics found in on-site soil samples. For example, in some on-site materials lead levels were measured at 93 to 1392 parts per million ("ppm") with a mean value of 367 ppm, and lead levels from two on-site road fill samples were 4190 and 4860 ppm. Lead levels in background soils, however, ranged from only 2.4 to 9.7 ppm with a mean value of 6.3 ppm. Inhalation of airborne particulates containing lead at the maximum concentrations found in on-site soils and other materials would exceed occupational standards for a continuous 8-hour exposure over a forty year period. The potential for the off-site migration of contaminants exists. Other than the removal of on-site drums containing hazardous substances, EPA selected the no action alternative with site access restrictions, the installation of two monitoring wells and one year of groundwater monitoring.*

2. *Highland Acid Pits, Texas R06-87/021. This site, 16 miles east of Houston, lies within the 10 year floodplain, has subsided 2.4 feet since 1964 and is bordered on two sides by the San Jacinto River. During the 1950's, the site received unknown quantities of industrial waste sludge believed to be spent sulfuric wastes from a refinery process. The primary groundwater contaminants are VOCs and heavy metals. These contaminants are present in an underlying shallow aquifer and have penetrated the region between this aquifer and a lower, middle aquifer. The ROD notes that the middle aquifer could become contaminated. Chromium has*

been detected in the San Jacinto River and/or other surface waters. EPA selected the no action alternative with long-term groundwater and surface water monitoring.

3. *Westline, Pennsylvania R03-90/086. This site, completely surrounded by the Allegheny National Forest and situated along the Kinzua Creek, was the location of a chemical plant that deposited tar material containing phenolic compounds and polynuclear aromatic hydrocarbons ("PAHs") into on-site lagoons and small canals, allowing the material to migrate downhill toward the creek. In 1983, EPA conducted an immediate removal of 2,000 tons of tar and contaminated soil. Although a 1986 ROD required the excavation and off-site incineration and disposal of an additional 2,340 tons of tar and contaminated soil, it did not address another estimated 4,000 tons of tar. EPA subsequently determined, based on an updated risk assessment using more recent risk criteria for PAHs, that the remaining tar and soil fail to pose a potential carcinogenic risk greater than the range of acceptable risks found at other Superfund sites. EPA amended the 1986 ROD discontinuing remediation requirements. As part of the no action alternative, the site will be monitored and reviewed again in five years.*

4. *M&T DeLisa Landfill, New Jersey R02-90/108. This 132 acre site includes a 39 acre area that was used as a landfill. A shopping mall exists on 30 acres of the former landfill. Landfill gas is generated at the site, and elevated VOC levels have been detected. Although EPA identified a potential human health risk from groundwater contamination under a future use scenario, EPA chose the no action alternative without even evaluating any remedial action alternatives. EPA transferred responsibility for the site to New Jersey with recommendations for the implementation of environmental controls including, inter alia, restricting possible future use of on-site groundwater, surface and groundwater monitoring, and periodic indoor and outdoor air monitoring.*

IV. EPA Unlawfully Biased the Community During the Public Comment Period

At the May 12, 1992 public meeting, a number of area residents and municipal representatives opposed EPA's Proposed Plan as a waste of funds. Some landowners also expressed fears of potential CERCLA liability as potentially responsible parties ("PRPs").

On May 24, 1992, an EPA employee appearing in a radio interview stated that EPA would release all landowners and municipalities and give them protection against contribution claims, all without any monetary settlement, in exchange for access to various locations within the Torch Lake site. The EPA employee stated that EPA would only seek monetary recovery from corporate PRPs.

Putting aside EPA's lack of authority to make such a commitment at this time, the bias that these statements reflect is both inappropriate and disturbing. Currying political favor without regard to law is a further demonstration of EPA's arbitrary and capricious conduct. We know of no precedent for such conduct.

We challenge EPA to explain how, under CERCLA, its representatives lawfully can attempt to influence public opinion in support of a proposed plan by proffering (during

the comment period) to certain PRPs covenants not to sue and contribution protection.

We further challenge EPA to explain the basis for its conclusion that it can lawfully make determinations as to covenants not to sue and contribution protection prior to issuance of the Record of Decision and any meetings with the PRPs in question.

Conclusion

The scientific data assembled as a result of EPA's studies show that the Torch Lake site is a safe, healthy and productive area. The data show that there is no meaningful risk to people or the environment from conditions there. The unsoundness of EPA's position is perhaps best illustrated by EPA's paradoxical and absurd statement that a soil and vegetation remedy is necessary to protect public health *unless the area in question is made into a public park.*

None of the justifications for remedial action under CERCLA is present at the Torch Lake site. The vast majority of the public in the area, as well as all local governmental officials, strongly oppose EPA's Proposed Plan. EPA should forthwith acknowledge that no remedial action is appropriate for Torch Lake and should forthwith delist the

site from the NPL. The data in the record permit no other conclusion.

Gaines Gwathmey, III

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Appendix to Comments Submitted
on Behalf of Universal Oil Products, Inc.
by Paul, Weiss, Rifkind, Wharton & Garrison

July 10, 1992

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on Behalf of Universal Oil Products, Inc.
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Document	Exhibit
Abelson, Philip, "Incorporation of New Science into Risk Assessment," Science, Dec. 14, 1990, vol. 250:1497	A
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Exhibit A

SCIENCE

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Incorporation of New Science into Risk Assessment

The Clean Air Act will eventually have some limited beneficial effect in reducing chemical risks to human health. It will be implemented at considerable expense to consumers. They will pay a subtle regressive tax, because industry will pass on to them increased costs. The act will have substantial hidden costs in creating numbing uncertainty in corporate planning and will probably lead to job losses in this country and weakened ability to compete globally. The act will enhance greatly the bureaucratic clout of the Environmental Protection Agency in its relations with industry. Principal beneficiaries of the act will be lawyers and entrepreneurial engineers.

Congress recognized that implementation of the bill will require enormous expenditures. Key members wished to be assured that the best science base available will be applied when costly standards are imposed. Their concerns were manifested in provisions in the act that stipulates, "The Administrator of EPA shall enter into appropriate arrangements with the National Academy of Sciences to conduct a review of . . . 'risk assessment methodology used by the Environmental Protection Agency to determine the carcinogenic risk associated with exposure to hazardous air pollutants. . . .' " The act also states, "In conducting such review, the National Academy of Sciences should consider . . . the techniques used for estimating and describing the carcinogenic potency to humans of hazardous air pollutants. . . ."

The study and report produced by the National Academy of Sciences could have consequences in other areas requiring risk assessment, including Superfund and the Resource Conservation and Recovery Act. Based on its current modes of risk assessment, EPA is embarked on programs that will cost hundreds of billions of dollars but will have little impact on human health. The questionable cornerstone of EPA policy is its dependence on studies involving administration of huge levels of chemicals to rodents and highly conservative modes of extrapolations to low doses in humans with the further assumption that at trivial doses a carcinogenic effect exists. The current guidelines select the most cancer-sensitive species as the yardstick despite the fact that it is known that biochemical and other processes often differ greatly between animal species and humans.

The NAS review is to be completed not later than April 1993. It is to be submitted to relevant congressional committees, to the administrator of EPA and to a new, high-level Risk Assessment and Management Commission. Three members are to be appointed by the President, six by leaders of Congress, and one by the president of the National Academy of Sciences. The act directs this commission to make an investigation of policy implications and appropriate uses of risk assessment in regulatory programs under federal laws to prevent cancer and other chronic health effects that may result from exposure to hazardous substances. The commission is directed to consider the report of NAS on risk assessment. The commission is also, among other things, directed to evaluate "the accuracy of extrapolating human health risks from animal exposure data. . . ."

The Clean Air Act also stipulates that the risk assessment report of NAS be considered by the administrator of EPA. Before taking certain actions "the Administrator shall publish revised Guidelines for Carcinogenic Risk Assessment or a detailed explanation of reasons that any recommendations contained in the report of the National Academy of Sciences will not be implemented."

Considerable evidence is already available that the standard EPA approach is outdated and more will be forthcoming as detailed studies of metabolic and physiological processes are made. Bruce Ames and his colleagues have produced substantial evidence that results of effects of huge doses of chemicals in rodents are often misleading. A major study at the Chemical Industry Institute of Toxicology has shown that carcinogenicity of formaldehyde is nonlinear; it decreases far more rapidly than dose. Studies on dioxin have shown that the high level of carcinogenicity manifested in some animals is of doubtful relevance to humans. Thirteen important substances including D-limonene (a constituent of citrus) and unleaded gasoline, cause kidney tumors in male rats but do not similarly affect other rodents or humans.

The EPA still sets guidelines on carcinogenic risks based on the limited information available during the 1970s. The agency needs to update its regulations as new facts are discovered. The study by NAS should lead to improved ways of identifying which substances are innocuous and which are truly dangerous and to better methods of making risk assessments in the light of scientific advances.—PHILIP H. ABELSON

Letters

When Kin Correlations Are Not Squared

In response to a number of inquiries concerning the proportion of genetic variance in IQ explained by the MZA [monozygotic] correlation, we have prepared the following explanation (Articles, 12 Oct., p. 223).

It is a common misunderstanding that the intraclass correlation is squared to estimate the proportion of variance explained by genetic factors. Familial correlations represent components of variance; they are not squared (*1*).

The reason that the intraclass correlation is not squared in our application is that the quantity to be estimated is the proportion of variance in twin A's IQ that is associated with twin A's genotype, and not the proportion of variance in twin A's IQ associated with twins B's IQ. In the latter case, an observed intraclass of 0.70 would be squared to yield an estimate of 0.49 for the proportion of IQ variance shared by the two twins. In the former case, however, the observed phenotypes are imperfect indicators of the underlying genotypes, so that the correlation itself provides a direct estimate of the proportion of IQ variance shared with the unobserved genotype. The situation is analogous to the estimation of reliability in psychometrics whereby the correlation between two parallel forms of a test provides a direct estimate of the proportion of observed test score variance associated with unobserved true score variance (that is, the reliability of the test) (*2*).

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REFERENCES AND NOTES

1. This result is best explicated by the use of a path diagram; see R. Plomin, J. C. DeFries, G. E. McClearn, *Behavioral Genetics: A Primer* (Freeman, New York, 1990), pp. 238–239.
2. This issue is also explicated by A. Jensen [*Psychol. Bull.* 75, 223 (1971)] from the point of view of reliability theory and the common elements formula for correlation. See, also, the reply to Jensen by J. K. Miller and D. Levine, *ibid.* 79, 142 (1973).

Frazil Ice

In the News & Comment article "Zebra mussel invasion threatens U.S. waters" by Leslie Roberts (21 Sept., p. 1371), reference is made to "frazzle" ice. "Frazil" is the correct spelling for the type of ice that blocked the Monroe, Michigan, water intake. This word, of French-Canadian origin, describes ice formed in turbulent, supercooled water. The term, from an Old French word meaning coal cinders (*fraisil*) apparently came into use because of the appearance of the ice.

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Imprisoned in Sudan

Moneim Attia, an eminent environmental physiologist from Khartoum, returned to Sudan some years ago after training and experience in Germany and Kuwait. His goal was to develop research on the problems raised by the local climatic challenges of his country. He was arrested in his home on the night of 13 January 1990. He has been detained without trial or accusation since then. We understand that his treatment has been inhumane in several ways, such as being kept without communication with his family, being frequently beaten, and being kept blindfolded day and night for long periods. His arrest was ordered by Lieutenant General Omar Hassan Al-Bashir, head of the Revolutionary Command Council for National Salvation, Khartoum, Sudan.

We the undersigned environmental physiologists urge our colleagues from all fields to write to Lieutenant General Al-Bashir, as well as to the ambassadors of Sudan in their countries, saying that they are aware of the bad treatment received by Moneim Attia and that this treatment (absence of trial or accusation, torture) violates several international conventions: (i) the Convention against Torture and Other Cruel, Inhuman or Degrading Treatment or Punishment; (ii) the Covenant of Civil and Political Rights; and (iii) the U.N. Body of Principles.

We understand that several other scientists are similarly detained in Sudan. What we do to defend Moneim Attia will have the general effect of helping protect all scientists who choose to help their countries.

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Carcinogenesis Debate

In her News & Comment article discussing our papers on carcinogens (9 Nov., p. 743), Jean L. Marx says that our position is, "Below the toxic dose, carcinogenesis would not be a problem . . . because there would be no increased cell proliferation," that is, thresholds are the general case. That is not our view, as is clear from our papers. It is reasonable to assume that low levels of mutagens might add a small increment to our enormous endogenous level of DNA adducts coming from oxidant by-products of normal metabolism. However, the risk should be considerably lower than predicted by linear extrapolation from high dose tests because increases in mitogenesis can be unique to high doses and inducible general defense systems act as a buffer at low doses. The risk from nonmutagens at low doses may be zero (for example, in the case of saccharin). Our view, as can be seen in our papers, is not that mitogenesis is a single-factor explanation for carcinogenesis. Rather our view is that you cannot understand mutagenesis (and therefore carcinogenesis) without taking mitogenesis into account and that at high doses chronic mitogenesis can be the dominant factor. This is also the view of S. M. Cohen and L. B. Ellwein and is supported by their work (Articles, 31 Aug., p. 1007).

Exhibit B

RISK ASSESSMENT ISSUES ASSOCIATED WITH CLEANING UP INACTIVE HAZARDOUS WASTE SITES

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ABSTRACT

Recent advances in risk assessment including more thoughtful approaches to ranking the weight-of-evidence for establishing the categories for carcinogens; biologically based dose-response modeling for carcinogens and other health end points; and the use of more accurate scientific information in exposure assessment are leading to refined outcomes from risk assessment. This paper surveys the background of risk assessment, including its conservative origins, and discusses the generalized approaches to hazardous waste site risk assessment as established by EPA's Superfund Manual. These approaches are compared to site assessments that can be developed using more advanced site specific data. To provide an enhanced scientific data base, far more attention must be focused on the risk assessment step of waste site remediation, i.e., the theoretical risk may be significantly less than the upper-bound risk established through the first screening exercise in risk

INTRODUCTION

The broad practice of using risk assessment approaches for the evaluation of suspected human carcinogens is about 12 years old. The primary departure point was the announcement by the U.S. Environmental Protection Agency (EPA) which adopted guidelines for assessing the risk of carcinogens and a policy to regulate suspect carcinogens based essentially on a risk management approach. The scientific basis was derived from the earlier experience of assessing the risk of health impacts from radiation exposure. From a practical standpoint, the use of risk assessment for carcinogens has received broad and general endorsement. The early use of risk assessment of carcinogens relied heavily on replacing the uncertainties in the risk assessment process with very conservative assumptions to make sure that, in no case, would the risk be underestimated. As the practice of risk assessment has become widespread, considerable attention has been focused on improving the scientific basis for evaluating each step of the risk assessment process: the weight-of-evidence indicating likely carcinogenicity, the dose-response relationships, and the environmental exposures. Chemicals which are though to cause health or ecological effects through threshold mechanisms are also being evaluated by risk assessment approaches. More attention has been focused on the scientific relationships that underlie the characterization of suspect carcinogens and their dose-response relationships including extrapolation from animals to humans and from high dose to low dose; far less attention has been focused on the exposure assessment which can impact the outcome of the quantitative risk assessment by certainly as much as the assumptions in the dose-response extrapolation part of risk assessment. Recent advances in both areas when applied to hazardous waste sites risk assessment, can substantially alter the outcome of the site-specific

risk assessments. This paper will provide an overview of scientific developments in risk assessment and describe how the use of these improved scientific data may alter the outcome of the standard superfund risk assessment approaches.

Risk Assessment: An Overview of the Process

In 1976, EPA adopted the first policy for the use of risk assessment of toxic chemicals which were suspected of being human carcinogens and accompanied this policy statement with guidelines for the scientific risk assessment process (EPA 1976; Albert et al. 1977). These guidelines were adopted in response to the need of a major regulatory agency to develop a means for regulating the presence of hundreds of suspected carcinogens in the environment under numerous environmental legislative statutes which had been adopted by Congress. In short, it was obvious that EPA could not regulate all suspected carcinogens which were being identified in rapid succession as environmental contaminants to a zero risk level as had been the risk goal of the Food, Drug and Cosmetic Act's Delaney Clause. Such a goal had clearly been the objective of the strong environmental movement which characterized the first half of the decade of the 1970s. The adoption of guidelines for risk assessment, with the implication that EPA planned to accept residual risk as a regulatory policy, was initiated under the watchful eyes of the scientific community, the regulated community, and the environmental communities. In short, there was considerable skepticism about the approach as a basis for public policy because of the substantial scientific uncertainties, particularly in quantitative risk assessment. After the EPA action, several other endorsements followed. The Inter-Agency Regulatory Liaison Group (IRLG) adopted similar scientific

principles in 1979 (IRLG 1979). These guidelines were followed by the report of the National Academy of Science which endorsed the use of risk assessment and provided descriptive terms for each step of the risk assessment process which have now been adopted as a common vocabulary (NAS 1983). In addition, the Office of Science and Technology Policy (OSTP) published similar scientific principles in 1985 (OSTP 1985) and EPA has updated its earlier guidelines (EPA 1986). In short, the application of risk assessment to toxic chemicals for the evaluation of scientific evidence that indicates that a chemical might be a human carcinogen and also provides information as to the magnitude of current and anticipated public health impacts has been endorsed and described in many forums and also has been the subject of discussion in many scientific conferences.

The process is generally described in four steps: hazard identification, dose-response modeling, exposure assessment, and overall risk characterization (NAS 1983). In practice, over the last 12 years, the hazard identification step in risk assessment has relied on all of the available human, animal, and/or in vivo or in vitro data to describe the weight-of-evidence that indicates that a chemical might be a human carcinogen. At various times, the weight-of-evidence has been stratified according to either the International Agency for Research on Cancer (IARC) criteria (IARC 1982) or the more recent EPA stratification scheme (EPA 1986a) for assigning a category to the weight-of-evidence. While these two categorical schemes are very closely related, the EPA scheme expands on the inadequate evidence labeled Category 3 in the IARC criteria to include three additional categories: C to indicate evidence that constitutes the category of "probable" carcinogen for humans; D to indicate inadequate testing; and E to indicate negative evidence.

Dose-response modeling has largely followed a linear nonthreshold hypothesis for low-dose extrapolation as a basis for defining a "plausible upper limit" on the risk meaning that the risks are unlikely to be higher but could be considerably lower (Crump et al. 1977; Crump and Watson 1979; Crump 1981; OSTP 1985; EPA 1986a). This model relies on the possibility that any suspected carcinogen can induce cancer by a single hit phenomenon and makes no distinction for different biologically based mechanisms of cancer induction. To date, other models which have variously been suggested for low-dose extrapolation from high-dose data have been empirically based models which seek statistically to define the best shape to the dose-response curve; they have not been based on data which seek to describe the biological events which lead to cancer.

Other assumptions, such as those used to extrapolate animal responses to humans, have been adopted which also have been chosen to be protective where scientific information was lacking (e.g., surface area is often chosen as the conversion factor rather than body weight). Dose is assumed to be synonymous with exposure, unless there are data to the contrary. Other conservative assumptions have also been chosen including, for example, the interpretation of the significance of benign tumors, which can lead to malignancy.

Exposure assessment likewise has followed a conservative trend. Generally, "maximum plausible levels" of chemical exposure have been used in risk assessments, sometimes in conjunction with "average exposure" estimates. An example of a frequently used conservative assumption is that an individual is exposed for a lifetime of 70 years unless there is evidence to the contrary. In practice, the overall risk characterization has relied on a ranking of the weight-of-evidence which placed considerable weight on any tumor response in animals and sought to quantitatively describe the risk to current or antici-

pated exposed populations as an "upper-bound risk" based on "maximum plausible" exposure estimates. The first decade of experience with carcinogen risk assessment has been studied both from the scientific standpoint and the use of risk outcomes in public policy decisions (Anderson and CAG 1983). In short, if scientists have been successful in the past describing risk assessment as "upper bound" estimates reflecting "maximum plausible exposures," then as better science is developed to fill the gaps of uncertainty, risk assessments should be expected to become less conservative.

Risk Assessment: Current Trends

Historically, protective assumptions replaced uncertainties; in some cases, uncertainty is now being replaced by improved scientific information. In the area of weight-of-evidence, fresh consideration is being given to the weighting of evidence at high dose and its appropriateness for low-dose weighting. For example, in the Carcinogen Assessment Group's risk assessment of ethylenethiourea (ETU) (EPA/CAG 1977), the uniqueness of the observation of rat thyroid tumors was discussed in the context of having a threshold, namely that these tumors resulted from suppression of thyroid activity only after the administration of a sufficiently high dose. Currently, the rat response is being examined to determine whether environmental exposure levels are likely to approach those that could be expected to elicit the rat thyroid tumor response; if not, it may be appropriate only to factor the mouse liver tumor response results into the weight-of-evidence determination for environmental exposure levels. Other chemicals are similarly being reviewed for their relevance to human exposure because of mechanism of action, tumor type observed, dosing

levels used, or metabolic and pharmacokinetic differences between humans and laboratory test animals.

The improvements in dose-response modeling probably represent the most dramatic departure from practices of the last 12 years. There is a clear effort by regulatory agencies to seek a biological basis for the development of more accurate estimates of risks expected to occur at environmental exposure levels. This effort represents a substantially different approach from applying empirical formulas to estimate low-dose responses from high-dose data; rather the attention is focused on the importance of research data that may guide low-dose modeling efforts. Such an approach provides, at a minimum, an indication of the extent to which the "plausible upper bounds" may be over-estimating risk for particular chemicals. Early efforts to define more accurate estimates of risk began at EPA in early 1985 and have culminated in the development of a generic approach using a two-stage model. This model adapts the clinical observations of Moolgavkar and Knudson (1981) to parameters involving exposure to toxic chemicals. The effort was first undertaken by EPA's Risk Assessment Forum and was ultimately published in the Journal of Risk Analysis in early 1987 (Thorslund et al. 1987). Thus far, EPA has proposed two important decisions in line with the trend toward less conservatism in dose-response modeling. Both of these decisions were discussed in a recent New York Times article (Shabecoff 1988). For example, the EPA's Risk Assessment Forum has recommended lowering the arsenic ingestion potency by approximately an order of magnitude (Levine et al. 1987; Moore 1987) based on modifications in dose-response calculation methodology and better estimates of the exposure involved in the epidemiology studies that were the basis for the evaluation. There is a further consideration of reducing the potency of arsenic by inges-

tion by still another order of magnitude to reflect the fact that skin cancer caused by arsenic ingestion is less likely to lead to death than is lung cancer induced by inhalation. Considerations of the latter raise the issue as to whether or not treatability, survival, and severity should be routinely considered as a part of the risk assessment process, and in particular the potency evaluation. In addition, EPA has proposed to downgrade the potency of dioxin based on several factors but most importantly, the use of the two-stage model of carcinogenesis for modeling the promoting activity of dioxin which indicates that the potency of dioxin may be two orders of magnitude or more less than the potency defined by the linear nonthreshold model for low doses (T.W. Thorslund and G. Charnley, in preparation). This work was prompted by recommendations of the EPA Science Advisory Board and is still under consideration (EPA/OPTS 1986b).

The two-stage model of carcinogenesis has also been applied to several other chemicals with similar outcomes. For example, the model has also been applied to chlordane and heptachlor, and methylene chloride (T.W. Thorslund et al. 1988, private communication). While the mechanisms in each case differ, the outcomes of the model are to indicate most often several orders of magnitude lower potency at low dose than predicted by the linear nonthreshold model at the "plausible upper bounds."

Additional applications of the biological model have involved the polycyclic organic compounds. Past practices have used the potency of benzo(a)-pyrene as a unit equivalency to all other potentially carcinogenic polycyclic organic compounds, greatly overestimating risk. This practice has continued in spite of the fact that comparative potency methods have been developed for other chemical classes, such as the dioxins. When assembled in the aggregate,

several laboratory studies provide a more substantial basis for developing a comparative potency approach for PAHs (M.M.L. Chu and C.W. Chen 1984, unpublished; Thorslund et al. 1986). In addition, the shape of the dose-response curve for benzo(a)pyrene itself has been reevaluated. Benzo(a)pyrene is a genotoxic agent as indicated by a linear rate of DNA adduct formation that parallels exposure. The tumor dose-response data do not parallel DNA adduct formation, however, but appear to fit a quadratic equation, indicating that two events are probably necessary to induce the response. EPA's initial cancer potency estimate for benzo(a)pyrene does not reflect this relationship. The comparative potency approach for other polycyclic compounds, together with a revised dose-response curve for benzo(a)pyrene, has been used to accurately predict tumor outcomes in bioassays of chemical mixtures, which is not possible using upper-bound estimates (Thorslund et al. 1986). Another example of a chemical which may require two events to produce a cancer outcome is benzene. Current investigations are examining the mechanistic data, which indicate benzene causes chromosome damage which is thought to be responsible for the chromosomal deletions and rearrangements observed in leukemia patients. This relationship implies that, although linearity may establish a plausible upper bound on human leukemia risk from benzene exposure, a quadratic relationship may be more appropriate to estimate the actual risk. Should this turn out to be the case, the risk from low dose exposure to benzene would be considerably lower than previously estimated (T.W. Thorslund and G. Charnley 1988, private communication).

A great deal of attention is also being focused on the metabolic and pharmacokinetic data to estimate actual levels of chemical exposure to the target tissue. In extrapolating animal data to humans, the effective dose in

the animal studies has always been assumed to be the dose that the animal was exposed to by route-administered-dose. As our ability to describe the actual dose to the target tissue in the animal improves, so will our ability to extrapolate animal responses to humans. In addition, the importance of pharmacokinetic data to define the significance of human exposure in the environment is exceedingly important.

Less progress has been made for threshold pollutants. While attention is currently focused on developing biologically based dose-response curves to better describe the threshold dose for disease causation, by the majority of these chemically induced effects are still described by applying safety factors to no-observed-effect levels (NOELs) from animals studies or for some few chemicals, describing the effective dose for observations in humans, e.g., lead. In either case, the results are uncertain and the outcomes subject to scientific debate.

Of equal importance, trends in exposure assessment research are also leading to improved estimates of population exposures which provide a better foundation for current and projected exposures. Traditional practices have relied heavily on generic models to describe exposure to human populations. EPA has developed generalized dispersion models for describing air transport and similar generalized dispersion models for surface and groundwater. The overall impact of these dispersion models has been to provide conservative estimates of exposure.

The use of generalized models provides a practical approach for widespread exposure estimation by regulatory agencies because it would be highly impractical for a national agency to evaluate site-specific parameters for every source. For important cases, however, it is possible to estimate actual

parameters that may refine the estimates obtained by generic modeling. An example is the risk assessment of the ASARCO smelter in Takoma, Washington which was conducted by EPA (Patrick and Peters 1985). The use of generalized dispersion modeling using the human exposure model (HEM) (which assumes a flat terrain, an immobile population, and uses meteorological data from the closest weather station), when coupled with the dose-response curve, estimated a maximum individual risk of about 1×10^{-1} for populations living near the smelter. Subsequently, a local study was conducted which permitted the use of several site-specific assumptions including a more accurate description of the actual terrain, local meteorological data, and better emissions information. The outcome was to lower the exposure assessment and the overall risk about an order of magnitude. This brought the risk into closer alignment with the limited monitoring data which was available for the ambient air.

The same phenomenon has been observed when comparing estimates using generalized dispersion models for groundwater with estimates which rely on site specific parameters. For example, in Figure 1, the generalized dispersion model, the verticle horizontal spread (VHS) model using EPA default values overestimates the risk by a factor of 5.7 when compared to the results from the more complex equation which incorporates measured site values (Domenico and Palciauskas 1982; EPA 1985). Another important area which has sharpened exposure estimates and practically has lowered the outcome from exposure assessment by several orders of magnitude and, thus the quantitative risk assessment, has been considerations of bioavailability. For example, dioxin was originally assumed to be 100% biologically available in soil. Recent studies, however, have demonstrated that dioxin is only partially available, >0.5%-85% depending on soil type (Umbreit et al. 1986). In practice, it has

been our experience that dioxin is mostly available in the range of 15-50% (P. Chrostowski 1988, private communication). Dioxin in fly ash also was originally assumed to be up to 100% available. Recent studies have found that this is not correct but rather that dioxin in fly ash is biologically available between 0.1% and 0.001% (van den Berg et al. 1986). The bioavailability issue is now being commonly investigated in many different situations where the availability in soil and fly ash is important to the outcome of the risk assessment.

Although improving the scientific information available for site-specific exposure assessment tends to lower the overall outcome of the exposure assessment and thus the risk assessment, there are important exceptions. For example, a recent paper which addressed the issue of risk associated with inhaling volatile organic chemicals from contaminated drinking water during shower activity (Foster and Chrostowski 1987) indicated that as much as half or more of the total body risk could be associated with the shower exposure rather than with the drinking water exposure. In addition, recent improved methods for modeling the actual deposition of particulate matter from stationary sources tends to raise the risk compared the earlier EPA air transport models which assumed that both large and small particles bounced from the surface of the earth in very similar ways and were carried from the site by air transport. The more recent models take into account that the small particles deposit on the surface and are not so readily transported (Sehmel and Hodgson 1979). Also, closer attention to chemical conversions may tend to raise or lower the risk; for example, trichloroethylene is converted under anaerobic conditions to vinylchloride which has a higher potency value by ingestion than does trichloroethylene (Parsons et al. 1984; Cline and Viste 1984). Recognition of this

conversion raises the overall risk assessment for circumstances which appropriately are evaluated by these methods.

Numerous other refinements in exposure assessment are also being incorporated in the risk assessment process, for example, use of human biological data to assist in exposure estimation, better descriptions of life style for subpopulation groups, the use of statistical methods to describe likely exposure below detectable limits, and the use of pharmacokinetic data to describe the actual dose to target tissue. These developments rely on advancing research in multiple disciplines for use in the practical consideration of human exposure.

APPLICATIONS TO HAZARDOUS WASTE SITE RISK ASSESSMENT

Wates site risk assessment practices have roughly paralleled the conservative (public health protective) approaches of risk assessment approaches over the last dozen years. The majority of this experience has been gained from the investigation of superfund sites according to the standard EPA Superfund Manual and related guidelines (USEPA 1985a, 1986c,d). In these investigations a risk assessment is a formalized methodology applied to determine the potential for human health and environmental impacts associated with a site under the no action alternative or to evaluate the potential benefits from remedial alternatives.

Generally, the initial step in conducting a risk assessment involves a review of all available site environmental monitoring data in order to select potential chemicals of concern on which the assessment will focus. At this step, chemical measurements with inadequate quality assurance/quality control or chemicals that are present as part of natural background may be rejected for

inclusion in the assessment. The next step, hazard identification, involves identifying chemical-specific human health and ecological effects criteria. This may involve an evaluation of available data, including epidemiology, animal bioassay studies, and in vivo and in vitro studies. In the absence of human data to describe low-dose effects, the frequently used approaches for dose-response characterization are for "threshold" (non-carcinogenic) and "non-threshold" (carcinogenic) effects. These approaches generate numerical health effects criteria to be used in the calculation of risk. While some guidance levels generally exist for most toxic chemicals, further scientific work may be warranted. Recent reconsideration by the USEPA of potency factors for arsenic, dioxin, and polycyclic aromatic hydrocarbons are good examples. Following hazard identification, potential pathways by which human populations may be exposed under current or potential future land-use conditions are identified. An exposure pathway is composed of the following four elements: 1) a source and mechanism of chemical release to the environment; 2) an environmental transport medium (e.g., groundwater) for the released chemical, and/or a mechanism of transfer of the chemical from one medium to another; 3) a point of potential contact of humans or biota with the contaminated medium (the exposure point) and; 4) an exposure route (e.g., ingestion) at the exposure point. All four of these elements must be present for a pathway to be considered complete. To evaluate exposure at an exposure point the concentration of chemicals of concern must be evaluated. Many times these are actual measured concentrations, however when they have not been measured, or to estimate future concentrations expected to occur over a longer time (i.e., a 70 year lifetime), or at exposure points not previously investigated, environmental fate and transport modeling may be necessary. For Superfund sites, once concentrations of

chemicals of concern at the exposure points have been determined, they are compared with "applicable or relevant and appropriate requirements" (ARARs). When ARARs are not available for all chemicals in all media, quantitative risk estimates are developed by combining the estimated intakes of potentially exposed populations (often derived using conservative assumptions regarding chemical concentrations, exposure duration, exposure frequency, and the efficiency of absorption in biological media of chemicals) with either existing health effects criteria or improved evaluations based on more recently available data and methods. Conservative assumptions are generally made in risk assessments to compensate for uncertainty and to explore the potential for adverse health effects using conditions that tend to overestimate risk so that the final estimates will usually be near or higher than the upper end of the range of actual exposures and risks. Greater uncertainty in the site-specific data base generally leads to more extensive reliance on conservative assumptions; conservative (i.e., protective) assumptions are chosen to make certain that risks will not be underestimated. Because there is uncertainty, risk assessments generally do not present an absolute estimate of risk; rather most risk assessments establish plausible upper bounds on risk to indicate the potential for adverse impacts. Thus, risk assessments are more useful where data are available to narrow uncertainties and permit the most accurate descriptions of risk possible. In the absence of such data, conservative approaches which provide upper bound risk estimates present clear guidance for the evaluation of low risk (i.e., that even at the upper bounds the risks are low and therefore most often do not warrant regulatory attention) but are less instructive for remedial prescription where the social and economic costs are high.

Potential Pathways of Exposure to Contaminants

All pathways of exposure are considered: groundwater, surface water, soil, and air. The pathway that is most often of greatest concern is groundwater. For purposes of discussion, this paper focusses on this route as an example of an exposure route evaluation.

To evaluate exposure to groundwater, standard intake assumptions are generally employed. These are that an average adult ingests 2 liters of water a day over a 70 year lifetime and that the average body weight over the exposure period is 70 kilograms, unless there are clear data to define alternative choices. For example, these assumptions can be arguably too stringent or in some cases, such as outdoor workers in an arid climate, they may not be stringent enough. If the demographics and activity patterns of the population are known, more accurate intake assumptions may be used which will often diminish risks.

Additionally, inhalation exposures to contaminants in groundwater may occur through use of water in day to day activities such as cooking, bathing, washing of dishes and clothes, or showering. Dermal exposures are also possible. Although many of these exposures may be dependent upon individual water-use patterns, exposure through showering may be quantified using the model of Foster and Chrostowski (1987). For many volatile organics quantification of the additional risks through inhalation of contaminants while showering may be similar to the risks associated with ingestion. In some instances, risks from all inhalation activities combined may be greater than those associated with ingestion, especially if the chemical involved is more toxic by the inhalation route (e.g., 1,1-dichloroethylene). Dermal exposures are

generally small compared to ingestion or inhalation, although they may be substantial when chemicals which are absorbed with a high efficiency (e.g., dimethyl sulfoxide) are involved. Failure to assess these pathways could lead to groundwater risk management decisions not protective of public health or associated with an inaccurate representation of liability.

In many locations groundwater discharges into surface water bodies create additional potential pathways of exposure. This is a particular concern for water bodies that are of moderate size (i.e. that have sufficient flow to support aquatic life and are not so large in volume as to dilute concentrations of contaminants discharging in groundwater to insignificant concentrations). Contaminants that have high octanol-water partition coefficients (K_{ow}s) have a potential to bioaccumulate and generally are of particular concern. These chemicals may not only be toxic to aquatic life but may potentially cause risks to other organisms higher in the food chain or to humans that ingest fish from these surface water bodies on a regular basis. Additionally surface water bodies may be used for other recreational activities such as swimming, or for drinking water supplies; these may create additional exposure pathways to contaminants in groundwater.

An added consideration is needed to provide an assessment of anticipated exposures. For example, a change in local pumping conditions due to the installation of a new well (particularly one that has a high yield such as an industrial or municipal well) may have an influence on contaminant migration or groundwater which is not currently a drinking water source may become a source in the future.

In evaluating potential exposure to contaminants present in groundwater, it is important to define the aquifer(s) to be evaluated. This may be a

tial source areas, thus conservative assumptions are generally made with regard to the nature, extent, frequency, and duration of chemical release to the subsurface.

Oftentimes estimation of concentrations of chemicals of concern in the groundwater begins with evaluating the transport of contaminants from the source, which is often soil, through the unsaturated zone. This can be done through a variety of approaches ranging from a simplified steady-state soil-water partitioning model to time dependent models that consider linear adsorption/desorption without accounting for dispersion (Enfield et al 1982) or more complex compartmental numerical models that incorporate time-varying transport, advection and dispersion such as the Pesticide Root Zone Model (USEPA 1984). Applying these models to the same site may result in soil pore water concentrations that may vary by as much as several orders of magnitude.

The output from the unsaturated zone models may then be coupled with groundwater models to predict concentrations of chemicals of potential concern at potential exposure points. The groundwater models may range from simplified mass-balance mixing models through analytical solutions of transport equations to complex three dimensional numerical models. In some instances simplified models used for screening purposes show that even using conservative assumptions the estimated concentration at a potential exposure point may not be associated with a risk and the conservative assessment may be sufficient. However refinement of conservative assumptions is often necessary to ensure that the evaluation is realistic and that remedial actions will not be undertaken needlessly.

An example of the importance of applying site specific parameters to a hazardous waste site in the context of groundwater solute transport modeling

can be instructive. For example, the Vertical Horizontal Spread (VHS) Model (USEPA 1985a, 1986f) is a steady state groundwater model in which the only attenuation mechanism is vertical and horizontal spreading; it neglects longitudinal dispersion, and chemical degradation kinetics. Application of the VHS model to a particular site using USEPA fixed default values resulted in a ratio of concentration at the exposure point to source concentration (C/C_o) of 0.34. However, a refinement of the model incorporating vertical and horizontal dispersion coefficients, and site specific parameters resulted in a C/C_o ratio of 0.06. Thus the generic USEPA model overestimated the concentrations and risks at the exposure point by a factor of 5.7 (Figure 1).

In many instances the steady-state assumption is not applicable. At another site, site specific groundwater modeling incorporating chemical decay and source decay illustrated that assuming that the observed concentration persists over 70 years could result in a substantial overestimation of the risk. For example, work completed by our scientist at a site in California indicated an "upper-bound" lifetime risk associated with ingestion of water containing trichloroethylene (TCE) in an aquifer to be as high as a 10^{-3} risk (Figure 2). This level is associated with a 70-year lifetime exposure via exposure to drinking water from the contaminated aquifer. Scenario 2 in Figure 2 describes the decline in risk associated when hydrogeology models are applied to the site; the model assumes that the source of contamination has been removed. In Figures 3 and 4 the monitoring well data are displayed and likewise the risk comparison over time given the ability to model the area. In this particular circumstance, remedial action was being considered which would cost in the million dollar range and require a number of months to install. If the hydrogeology models are correct, the theoretical risk could be lowered con-

siderably over the first 18-month period given the natural ability of the hydrogeology of the area to remove the contamination. Caution, however, should be exercised in assuming that the source has been removed because recent publications indicate that in some circumstances some chemicals may remain entrapped in soil micropores and thereby provide a slow, diffuse release (Sawhney et al. 1988).

As the concentration of a particular compound present in groundwater may decrease through natural processes such as biodegradation, the total risk resulting from exposure to the groundwater may not necessarily decrease. Some compounds are transformed into more toxic compounds through biotransformation. For example, under anaerobic conditions, some halogenated aliphatics have been found to undergo reductive dechlorination (Bouwer et al. 1981, Kobayashi and Rittmann 1982, Vogel and McCarty 1985). The transformation is sequential, with, for example tetrachloroethylene yielding first trichloroethylene (TCE) and, ultimately vinyl chloride (Parsons et al. 1984, Cline and Viste 1984). Trichloroethylene is categorized as a probable human carcinogen with a cancer potency factor of 0.011 $mg/kg/day^{-1}$ whereas vinyl chloride is a known human carcinogen with a cancer potency factor that is approximately two orders of magnitude greater than TCE by the ingestion route. Biotransformation is dependent upon a variety of factors such as availability of organic chemicals, oxidation/reduction conditions, availability of microorganisms, growth factors, toxicity, and inhibition. Consequently the extent to which biotransformation may occur in groundwater at a particular site will vary and the degree to which it occurs is difficult to quantify. Use of a risk assessment for TCE without considering the potential impact of vinyl chloride could result in an underestimation of risks at the site. This could lead to risk management decisions

from an inaccurate data base which could be ultimately expressed as lingering liability even after cleanup had been accomplished.

Summary

In summary it has been shown that risk assessments are generally conservative evaluations primarily due to selection of assumptions to compensate for data limitations and uncertainties. The methods selected to estimate exposure and to quantify exposure point concentrations may have a substantial effect upon the estimation of risk associated with exposure to contaminants in groundwater. Most often more accurate data will provide risk assessment outcomes that are less conservative; though use of the shower model and chemical transformation to more potent chemicals can have the opposite impact. There is no question that the best hazardous waste site clean up decisions must rely on the most accurate risk assessments possible; thereby stressing the importance of accurate initial site characterization. The implications of overestimating potential risks associated with contaminants present in groundwater or other media may result in implementation of expensive remediation that could have otherwise been less restrictive. Conversely, inaccuracies in predicting risk may also result in underestimations of exposure which may have far reaching ramifications in the areas of public health protection and liability evaluation. Currently fare more attention is focussed on costly remediation than on reducing the theoretical risk through better risk assessments. Substantial experience demonstrates that improved risk characterization is possible for most sites. This additional scientific effort is important to the process of distinguishing which sites require, the greatest attention for remediation.

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Ground Water Modeling

1. VHS model using simple equation, EPA fixed default values

$$\frac{C}{C_O} = \text{erf}\left[\frac{Z}{2(DY)^{0.5}}\right] \text{erf}\left[\frac{X}{4(DY)^{0.5}}\right]$$

$$= 0.34$$

2. VHS model using complex equation, measured site values

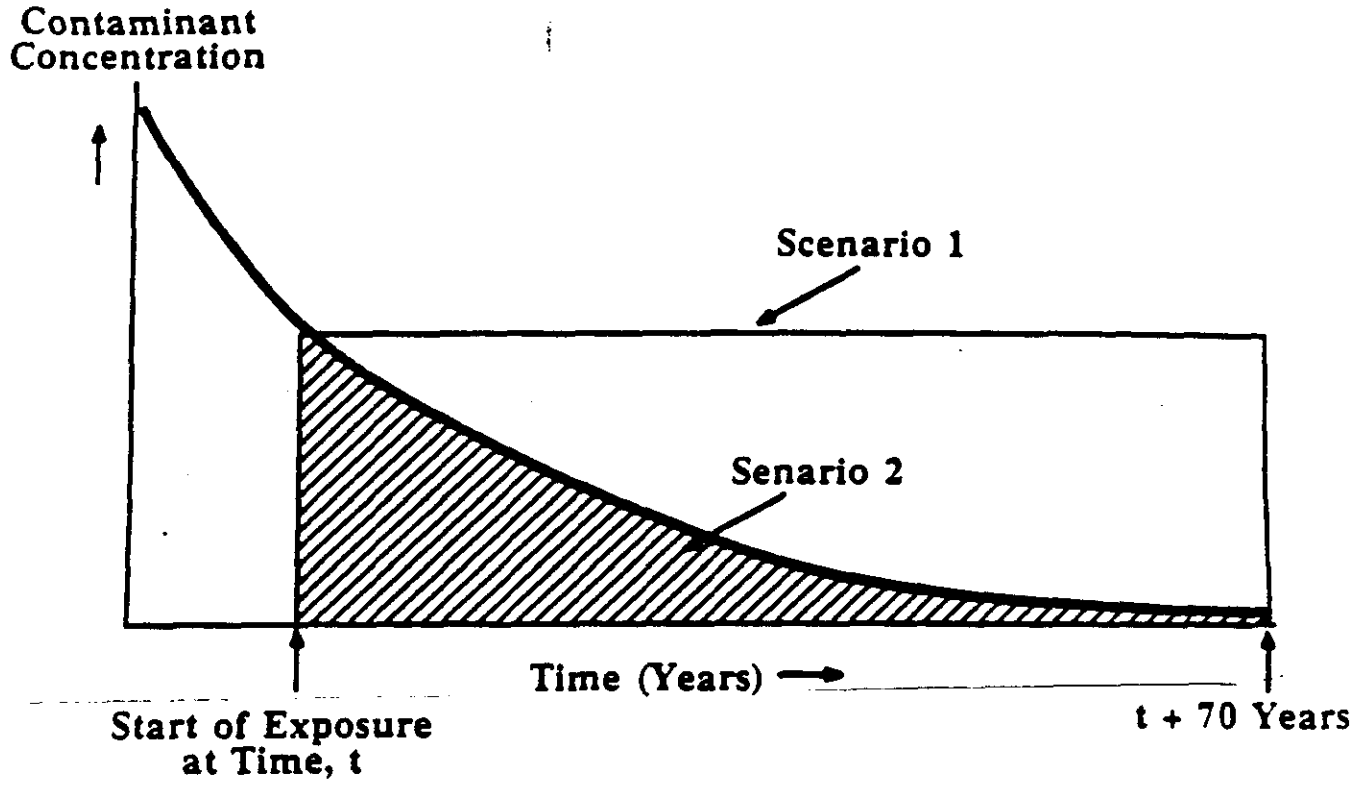
$$\frac{C}{C_O} = \frac{1}{4}\left[\text{erf}\left(\frac{z+Z}{2(D_zY)^{0.5}}\right) - \text{erf}\left(\frac{z-Z}{2(D_zY)^{0.5}}\right)\right]$$

$$\left[\text{erf}\left(\frac{x+X/2}{2(D_xY)^{0.5}}\right) - \text{erf}\left(\frac{x-X/2}{2(D_xY)^{0.5}}\right)\right]$$

$$= 0.06$$

Conclusion: EPA method overestimates concentration at exposure point by factor of 5.7.

FIGURE 1



Scenario 1 assumes decay in contaminant levels until start of consumption and then a lifetime exposure to a constant concentration of the contaminant.

Scenario 2 assumes decay in contaminant levels both before and during the period of exposure.

FIGURE 2

GROUNDWATER MONITORING DATA

Well #	Sampling Date	[TCE] mg/l		Well Average [TCE] mg/l
1	1/1/85	.100	.124	
	1/20/85	.144		
	2/2/85	.127		
2	1/1/85	<.005	1.27	
	1/20/85	1.10		
	2/2/85	1.98		
	2/8/85	2.00		
3	1/1/85	<.005	.003	
	2/2/85	<.001		
4	1/1/85	.055	.068	0.36
	2/2/85	.080		
5	1/20/85	.210	.194	
	2/2/85	.177		
6	1/1/85	.510	.96	
	2/8/85	1.40		
7	1/20/85	.160	.23	
	2/2/85	.305		
8	1/1/85	.070	.04	
	2/8/85	<.005		

FIGURE 3

Time of Exposure Initiation (Years)	[TCE] at Start of Exposure (mg/l)	Lifetime Upper Bound Cancer Risk	
		Scenario 1	Scenario 2
t_0	.36	1.1×10^{-4}	3.5×10^{-6}
1	.23	7.1×10^{-5}	2.2×10^{-6}
2	.14	4.5×10^{-5}	1.4×10^{-6}
3	.09	2.8×10^{-5}	8.8×10^{-7}
4	.06	1.8×10^{-5}	
5	.04	1.1×10^{-5}	
6	.02	7.2×10^{-6}	
7	.01	4.6×10^{-6}	
8	.009	2.9×10^{-6}	
9	.006	1.8×10^{-6}	
10	.004	1.1×10^{-6}	

FIGURE 4

Exhibit C

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INSIDE

default value for this factor is 2 liters of tap water consumed per day. As it turns out, this is approximately the 95th percentile of tap water consumption in the U.S. population (i.e., only five percent of the U.S. population consumes on average more than 2 liters of tap water per day).

Early on, the Superfund program codified many standard assumptions on exposure and dose-response assessment in two core guidance documents: EPA's 1986 *Superfund Public Health Evaluation Manual* (SPHEM) and 1988 *Superfund Exposure Assessment Manual* (SEAM). Then, with the 1989 publication of the *Exposure Factors Handbook* (EFH), EPA adopted even more standard factors for assumptions about the body weight of individuals, the volume of water ingested by children and adults, the mass of soil inadvertently ingested by children outdoors, etc. By and large, risk assessors found these reports useful, because they provided extensive information condensed into a few volumes and because they allowed risk assessors to substitute site-specific information as appropriate in particular analyses.

In late 1989, EPA updated its risk assessment guidance by publishing *Risk Assessment Guidance for Superfund, Volume 1, Human Health Evaluation Manual, (Part A)*. This volume (commonly referred to as RAGS, or HHEM) contained even more standard exposure factors, and the Agency began an effort to make new standard exposure factors even more conservative (i.e., protective of public health).

To counterbalance the tendency of many regional and headquarters staff members to require the concatenation of a series of conservative assumptions in a risk assessment, the Agency formally adopted the concept of the RME as explained in these excerpts from Section 6.1.2. of RAGS (with the emphasis in the original):

> Actions at Superfund sites should be based on an estimate of the *reasonable maximum exposure (RME)* expected to occur under both *current* and *future* land-use conditions. The reasonable maximum exposure is defined here as the highest exposure that is reasonably expected to occur at a site.... Estimates of the reasonable maximum exposure necessarily involve the use of professional judgment.... The intent of the RME is to estimate a conservative exposure case (i.e., well above the average case) that is still within the range of possible exposures.

In addition to this general discussion of the RME concept in RAGS, the Agency elaborated on its definition of "reasonable worst case" (which we equate to the RME) in Appendix 2 of the EFH. In this document, EPA discussed the reasonable worst case as exposures in the 90th to 95th percentile range. Although it had no official position on how to define the reasonable worst case exposure scenario, the Agency recommended using "a combination of some lower values [50th percentile] and some upper values [90th to 95th percentile]." Furthermore, the Agency concluded that the "best" approach for deriving the reasonable worst case exposure level was by using Monte Carlo techniques, a statistical procedure that we discuss in more detail later.

Thus, it seems unmistakable that EPA *intended risk assessors to utilize procedures* that lead to reasonable estimates of risk that no more than five to ten percent of the *population exposed would likely exceed.*

These recent Superfund guidance documents clearly demonstrate the Agency's understanding that the combination of a series of conservative assumptions can yield an overall estimate of risk that has little or no meaning in reality. Unfortunately, the Agency's recommendations on the choice of default exposure factors in RAGS do, in fact, lead to the use of a series of conservative assumptions yielding risk estimates that are highly unlikely to be experienced by a typical population surrounding a Superfund site.

To improve consistency in the use of the Agency's risk assessment guidance across its ten regions, the Office of Solid Waste and Emergency Response (OSWER) issued a directive on March 25, 1991, that most directly and clearly subverts its previous policy decision that risk assessments be based on reasonable exposures (90th to 95th percentile). The crux of this directive is contained in the first two pages:

> This supplemental guidance attempts to reduce unwarranted variability in the exposure assumptions used to characterize potentially exposed populations in the baseline risk assessment. This guidance builds on the technical concepts discussed in HHEM [RAGS] Part A and should be used in conjunction with Part A. However, where exposure factors differ, values presented in this guidance supersede those presented in HHEM Part A (page 1).
>
> These standard factors are intended to be used in calculating reasonable maximum exposure (RME) estimates for each applicable scenario for a site. Readers are reminded that the goal of RME is to combine upper-bound and mid-range exposure factors in the following equation so that the result represents an exposure scenario that is both protective and reasonable, not the worst possible case

$$Intake = \frac{C \bullet IR \bullet EF \bullet ED}{BW \bullet AT}$$

where

C = Concentration of the chemical in each medium (conservative estimate of the media average contacted over the exposure period)

IR = Intake/Contact Rate (upper-bound value)

EF = Exposure Frequency (upper-bound value)

ED= Exposure Duration (upper-bound value)

BW= Body Weight (average value)

AT= Averaging Time (equal to exposure duration for noncarcinogens and 70 years for carcinogens) (page 2)

Thus, of six variables in the standard exposure equation, EPA now requires risk assessors to use upper-bound values for three. Generally, EPA uses the phrase "upper-bound value" to refer to the 95th percentile of the full risk range. Using a simple relationship from probability, the multiplication of three 95th percentile numbers (assuming log-normal distributions) yields a value close to the 99.8th percentile which is just shy of "3 nines" conservatism. In other words, the Agency's operational definition of the RME means that at this exposure there would be less than 1 chance in 500 that an individual exposed to a Superfund site would receive exposures greater than the RME.

This "3 nines" conservatism in the exposure calculation can be further compounded if the Cancer Potency Factor (CPF) for a chemical is derived by similarly conservative methods, since the lifetime cancer risk is ultimately derived by multiplying the calculated exposure by the CPF. Derivation of the CPF has probably been the longest standing controversy within the scientific risk assessment community. Adding another conservative value to the risk equation to represent the toxic potency of the compound would only make the point estimate of risk less plausible.

For carcinogens with CPFs based on animal data, EPA derives the CPFs for humans by extrapolating from the upper 95th percent confidence limit on the linear term of a curve-fitting model (called the linearized multistage model) that is used to fit the data for the most sensitive sex of the most sensitive laboratory test species (usually rats or mice). Not only can derivation of the CPF be exaggerated by the curve-fitting model that is used to extrapolate data from high-dose animal experiments to low-dose human exposures typical of most Superfund sites, but the experimental protocols used in testing laboratory animals can also bias the results in the direction of overestimating cancer risk. This can be especially true for chlorinated hydrocarbons, a class of chemicals ubiquitous at Superfund sites that often drive cleanup decisions, especially for ground water contamination. One chlorinated hydrocarbon, chloroform, was recently retested in animals using more appropriate laboratory protocols than those first used by the National Cancer Institute in the mid-1970s; the results led EPA to lower the CPF for chloroform by a factor of 10. Although there are theoretical situations where the opposite may be true, the point is that EPA commonly uses procedures to derive CPFs from animal data that are designed to add more conservatism to risk assessments.

EPA's specification of simplistic assumptions in its guidance documents has a deeper and more unfortunate side effect—it arrests further inquiry into the subject at hand. Once the Agency announces a simple assumption, neither risk assessors nor the Agency itself has an incentive to research the topic for which the assumption was used to fill the gap in knowledge. For example, with NRC's publication of the first *Drinking Water and Health* report in 1977, the Safe Drinking Water Committee of the NRC/NAS adopted the value of 2 liters/day as the amount of tap water a typical adult ingests in a day in food and beverages. While the Committee offered little justification for this number, EPA has adopted the value in many programs and regulations, apparently with little further inquiry. EPA has had no incentive to look for data on the topic, nor have they encouraged the regulated community to look beyond the simple policy. Only recently did an alternative appear, when Roseberry and Burmaster (1990) analyzed data for the daily volume of tap water ingested by five age groups that were compiled by scientists evaluating the Nationwide Food Consumption Survey conducted by the U.S. Department of Agriculture in 1977 and 1978. Although these data can be easily used to improve the quality of risk assessments, most EPA regions have blocked the use of any drinking water factor other than 2 liters/day.

Risk Assessment Today at EPA's Regional Offices

With the exception of Region III in Philadelphia, most risk assessors in many of EPA's regional offices have a record of interpreting national guidance in a more conservative manner than its already conservative intention. We are aware of many situations where regional staff have argued that a risk assessor should—indeed, must—combine the 90th or 95th percentile value for *each* of five or six variables in each exposure pathway, which has the effect of driving the risk estimate well beyond the 99.99th percentile estimate.

In many cases, EPA regional staff require the use of exposure factors that border on the absurd. For example, citing the most recent OSWER directive, risk assessors in Region I assume that children could inadvertently ingest 200 mg of contaminated soil each day for 350 days/year, even in northern Maine where snow and ice cover the ground for many months each year. The soil ingestion rate for children was derived from studies of day-care children playing out of doors during the warm summer months. EPA's conservatism in assuming that children ingest these quan-

tities virtually every day of the year is based on the uncertainty as to how much indoor dust children ingest during inclement weather, and the extent to which this dust may contain contaminants from the Superfund site. This same assumption is often used even for children who may inadvertently come in contact with the Superfund site, yet live at locations remote from the site where indoor dust may be affected little by the site.

Similarly, in draft but binding guidance, the Region I Office of EPA in Boston requires risk assessors to use the maximum reported concentration of each chemical in a risk assessment. Thus, for an exposure scenario that considers inadvertent soil ingestion, in effect, Region I forces risk assessors to assume that *all children (1) have perfect knowledge of the location on the* site with the highest surface concentration of each chemical, and (2) wander from location to location, sometimes hundreds of feet apart, *to ingest soils containing the maximum concentration of* each chemical. At a Superfund site in Region V, Agency risk assessors went a step further and assumed that the hypothetical child would also dig through a multimedia cap (5 feet of clay and two plastic liners) to reach the contaminated soil below. Such policies have no scientific merit and clearly drive remedies to the extreme.

A Path to Disentangling Risk Assessment from Risk Management Decisions

Short of restoring reasonable professional judgment to EPA risk assessors, there is no methodological "quick fix" to many of the important assumptions governing the assessment of health risk at Superfund sites, such as the intended future use of the site. There is, however, a way to reintroduce science and fact into risk assessment calculations without biasing the results with implicit risk management decisions.

The Monte Carlo method provides a statistical methodology for separating the risk assessment process from risk management decisions. It provides a methodology for estimating probability distributions for exposure and risk, and it avoids a prolonged dispute on how to estimate the most appropriate point estimates of exposure and risks. It also maximizes the information content of the risk assessment and provides the risk manager with a wider array of options from which to choose. For example, a Monte Carlo simulation not only presents the risk estimates as a probability distribution (e.g., the risk can be estimated for any percentile of exposure), but the extent to which the risk estimates are sensitive to the uncertainties surrounding the input variables (e.g., the quantity of water consumed/day) can be easily explored. Thus, not only can risk managers define "how clean is clean" in the context of the uncertainties of the risk assessment, they can also specify additional investigations aimed at factors that contribute most to those uncertainties.

In fact, the Monte Carlo method, sometimes called probabilistic risk assessment, starts with the realization that it is impossible to assess risks in terms of any single number unless that number is associated with an estimate of the probability that any individual in the population will experience that risk. It is a method that explicitly links risk (e.g., a lifetime cancer risk of 10^{-6}) with the probability that any member of the public will experience that risk, or exceed it. We believe that it is extremely important to convey the overall probability distribution on the estimated risk in an unbiased way to risk managers and the public. As a society, we must make decisions based on unbiased science and the full presentation of the range of possible outcomes, not on a set of counterfactual assumptions. Even more, we need to unfetter our risk assessors so that they can pursue estimates of risk by the use of objective science and fact, not just by repeating simplistic Agency policy.

With desktop computers as powerful as mainframes were just a few years ago, analysts can now use relatively simple commercial software to estimate full probability distributions —not just point estimates—for health risks experienced by populations chronically exposed to toxic chemicals from hazardous waste sites. Even though probability is the central concept in risk assessment, and even though probabilistic methods offer strong advantages and insights as compared to the deterministic methods now required by EPA's guidance manuals and directives, analysts have only recently begun to use Monte Carlo methods at Superfund sites.

Monte Carlo simulation, developed by physicists over 50 years ago and long used by engineers in many fields, addresses the weaknesses of the current risk assessment methods embodied in EPA's guidance documents. In Monte Carlo simulation, each of the many input variables (e.g., daily volume of drinking water consumed, years of residence at any one location) can become a random variable with known or estimated probability distribution. Within this framework, an exposure variable takes on a range of values with a known probability. In extending the regular methods used in public health and ecological risk assessments, probabilistic techniques add several steps to estimate both point values and full probability distributions for the exposures and risks.

First, the analyst determines a continuous or discrete probability distribution to describe each of the exposure variables to be included in the analysis, although point estimates can also be used if the underlying probability distribution for a variable is not known or cannot be reliably estimated. In this step, the analyst must also determine if any correlations exist among the input variables and account for them if they do (e.g., body surface area is correlated with body weight and this correlation must be accounted for in the analysis). Second, the analyst uses suitable computer software to make a large number of risk calcu-

lations. For each calculation, the computer selects one random value from the appropriate probability distribution for each of the exposure variables in the model, and computes and stores the result of the risk calculation. This computation is repeated thousands of times. Third, the analyst establishes the shapes of the distributions for intermediate and final results and produces various statistical summaries of the results. In this framework, a complete risk probability distribution is derived, providing maximum information to the risk manager and members of the public. For example, if the collective judgment of the public and the regulatory agency is to base the cleanup decision on a level of risk with a less than five percent probability of being experienced by any member of the population, this could be uniquely provided by the Monte Carlo simulation.

To illustrate the differences between the results of point estimates using EPA's guidance and the same risk calculations using Monte Carlo simulation, we have estimated the lifetime cancer risk for a hypothetical case in which workers at an industrial facility are assumed to be exposed to a single carcinogen, chloroform, through a single exposure pathway, incidental ingestion of soil contaminated with chloroform at a concentration of 4 ppb throughout their working lifetime at the site. In this example, a point estimate of the lifetime cancer risk was calculated based on a risk-based method used by the New Jersey Department of Environmental Protection and Energy (NJDEPE) for developing proposed cleanup standards for industrial soil. NJDEPE's risk calculations are consistent with OSWER's recent guidance that requires the use of "upper-bound" values for the three exposure factors, IR (soil ingestion rate), EF (exposure frequency), and ED (exposure duration). This point estimate was then compared with the 95th percentile cancer risk estimate calculated using Monte Carlo simulation.

The following table contrasts the 50th and 95th percentile estimates of these three exposure factors for industrial workers (taking into account vacation time, sick leave, and inclement weather) with the upper-bound values currently being used by NJDEPE.

Exposure Factor	(percentile) 50th	90th	95th	State Agency
IR (mg/day)	10	50	89	100
EF (days/year)	126	167	183	245
ED (years)	4	19	29	25

As this table shows, NJDEPE chose exposure factors for IR and EF that exceed the 95th percentile estimates, and chose a value for ED that is between the 90th and 95th percentile estimates. Arguably, all three qualify under EPA's definition of upper-bound estimates. Using EPA's recommendations that upper-bound values be used for the three exposure factors results in a point estimate for the lifetime cancer risk of 8.3 per million, which exceeds the tolerable risk of one in one million established for chloroform in soil by NJDEPE. However, using Monte Carlo simulation results in a 95th percentile risk level of 9.5 per 10 million, almost one-tenth the risk calculated by the standard EPA method, and slightly below the tolerable risk of one in one million.

Clearly, this simple comparison suggests that if the risk management decision is to base regulatory decisions on 90th to 95th percentile exposures, the EPA-proposed method and Monte Carlo simulation can lead to very different conclusions. Furthermore, these differences can become even more exaggerated when there are more than three exposure variables, which is usually the case in Superfund risk assessments. In a recent example, we evaluated the risks associated with leakage from a landfill; the exposure model included 19 variables and the point estimate using upper-bound values for five of the 19 variables resulted in risks about 500 times higher than the 95th percentile estimate from Monte Carlo simulation.

Concluding Observations and Recommendations

An unfortunate trend—created in the name of policy consistency—has replaced the science in risk assessment with simplistic policy assumptions that have the effect of making risk assessments ever more conservative. Too many risk assessors with advanced degrees in toxicology, chemistry, engineering, and other technical disciplines are being forced to use (and to defend in public) contrived and biased methodologies to conform to EPA policy guidance. These methodologies simply cannot be defended as good science, and they subvert the Agency's stated risk management policies aimed at protecting the public health against *reasonably* expected future exposures at Superfund sites.

EPA would be well-advised to endorse and encourage the use of Monte Carlo simulations as a way of supplementing or replacing current Agency risk assessment methodology. Monte Carlo methodologies, and variants of them, can help to overcome some of the methodological flaws that have characterized the risk assessment process under Superfund by enabling the calculation of risks that flow from a statistically defensible estimate of the reasonable maximum exposure. This will help separate risk assessment from risk management decisions in the sense originally recommended by the National Research Council/ National Academy of Sciences and Mr. Ruckelshaus. With a return to the original intent, risk assessors can do their jobs in a reasonably unbiased way, and risk managers can make explicit decisions on "how clean is clean" in the form of "how conservative is conservative enough." In the process, some credibility will be restored to the Superfund program, and, ultimately, cleanup decisions will be more defensible. ■

Quantitative Environmental Analyses in Insurance Litigation

By Charles F. McLane, Ph.D., Manager

Litigation concerning contamination of environmental media has increased greatly within the past decade. Much of this litigation stems from insurance disputes that involve claims for damages arising from investigation and remediation of toxic chemical releases. ENVIRON has assisted in many of these cases in a technical role.

The issue in these cases is not whether a release event has occurred. The volumes of data routinely collected for soil, ground water, surface water, and air at the site of a release make clear the nature and extent of the contamination. Rather, the issues concern clauses in the insurance contract (or contracts in the case of multiple insurers) that form the focal points of dispute between insurer and insured. Typically, these clauses state that coverage is excluded if the discharge of chemical is not "sudden and accidental" or if only "owned property" is impacted. There are also "trigger" clauses that describe the time at which a covered "occurrence" is considered to have taken place.

Reconstructing the history of one or more releases from one or more sources and the subsequent movement of chemicals through various environmental pathways often can be difficult. Records of discharge are rarely kept and many releases from underground piping or storage tanks go undetected for years before the damage is discovered. ENVIRON has found, in many cases, a combination of historical records, recent site media sampling data, and the use of computer models of chemical fate and transport in the environment can be used to support or refute claims in insurance litigation matters. This article will discuss the ways in which computer modeling can be used to provide valuable information in environmental insurance disputes.

Key Insurance Litigation Issues

Four key issues arise in insurance cases. Two of these issues question (1) whether the costs incurred by the claimant represent damages covered by the policy or equitable relief not covered under the contract, and (2) whether the discharge was intended or should the release have been expected. The first of these issues is a matter of legal interpretation and, therefore, is not amenable to analysis through the use of computer modeling. The second deals with state-of-the-practice issues, usually handled by the testimony of experts with historical knowledge of storage or disposal practices, although engineering design calculations sometimes are valuable.

Computer models are often used, however, to provide analyses of the other two issues that commonly arise in insurance litigation: (3) whether the release of chemical was sudden and accidental, and (4) whether there is a potential for impairment of property other than the "owned property" of the insured.

The third issue deals with the initiation of the release, its duration, and its characterization as a single or repeated release, especially with respect to the "trigger" for the claim. Computer modeling can reconstruct the release based on the current pattern of contamination provided by sampling data and information on the key fate and transport processes. Data on possible release dates and volumes are always helpful but, as one of the case studies below illustrates, release scenarios can be reconstructed in the absence of such data.

Similarly, the "owned property" issue in a broader sense deals with questions such as the time the contamination first reached ground water (often considered to be the property of the state); the time at which contamination first extended off the owner's property or its potential to do so; and the actual or potential impact of the contamination on off-site receptors. The case studies presented below represent two of the many cases in which ENVIRON has used fate and transport modeling to reach successful resolution of insurance litigation disputes. Details have been modified slightly in each case to protect client confidentiality.

ASSESSING RISKS OF NEUROTOXICITY

Over a thousand years ago, excessive exposure to lead was known to cause convulsions in humans; in the last decade, we have become concerned about more subtle effects (for example, impaired intellectual functioning in children) that may be associated with very low exposures to lead. Today, concern is growing that other chemicals have the potential to injure the human nervous system, and neurotoxicity is an increasingly important component of a chemical's toxicological profile.

by Mary Burr Paxton, Ph.D., and Thomas B. Starr, Ph.D., Principal

Magnitude of the Problem

The complex nature of the nervous system and its functions means that it can be injured in a large number of ways. Injury can occur following acute or chronic exposures to both synthetic or naturally occurring materials at any time from conception to old age. The effects may be reversible or permanent, or lesions may not become evident until an individual experiences other stresses. (Some scientists now think an accumulation of small insults of this sort might explain Alzheimer's, Parkinson's, and other degenerative neurological diseases.) Neurotoxic damage may appear in an enormous variety of ways (structural, functional, sensory, behavioral, or cognitive) that may be difficult to distinguish from "background" variation; chemically induced behavioral changes may be particularly difficult to distinguish from normal behavioral responses to changes in social or personal factors. The health consequences of exposure to a neurotoxicant may also occur indirectly, as is clear from the all-too-frequent results of combining alcohol and driving; similarly, exposure to neurotoxicants in the workplace can pose a threat to general safety. Dr. Kent Anger reviewed 588 chemicals that the American Conference of Governmental Industrial Hygienists has listed on the basis of their general toxicity. He found that neurotoxic effects were one of the underlying reasons for establishing recommended exposure limits for 28% of them. Thus, neurotoxic effects may be the "most sensitive effect" for many chemicals and, therefore, would be a focus of their health risk assessments. Neurotoxicity would thus play a critical role in determining how these chemicals will be regulated.

The need for more routine and comprehensive neurotoxicity testing has received greater attention in recent years. A recent report from the Office of Technology Assessment, *Neurotoxicity: New Developments in Neuroscience,* noted that regulatory agencies have the authority to require neurotoxicity testing for existing and newly developed chemicals, but have been slow in exercising this responsibility or even defining what would constitute appropriate testing for such effects. The National Research Council is also expected to publish an Agency of Toxic Substances and Disease Registry-sponsored study on neurotoxicity testing and risk assessment in the near future.

Both the Food and Drug Administration (FDA) and the Environmental Protection Agency (EPA) have been revising

Although this simple and generic approach seems to have worked well to date, many people are dissatisfied with it and think it could be significantly improved. They raise these major objections:

- it is strictly empirical and lacks a theoretical basis;
- it ignores the slope of the dose-response curve in the vicinity of the NOAEL;
- it effectively discards all but a single data point; and
- it may be inappropriately sensitive to sample size.

Applying the usual safety or uncertainty factors to a statistically derived "benchmark" dose would circumvent some of these problems. This method still falls short of what could and should be done to perform low-dose extrapolation scientifically, because such "benchmark" doses have no substantive mechanistic basis.

Because the ultimate purpose of investigating the dose-response relationship is to gain insight into the likely response at exposures below those already observed, the utility of establishing a dose-response relationship by fitting general-purpose functions to the available data comes into question immediately. As assessors of carcinogenic risks have learned, different mathematical models that fit data equally well can diverge by orders of magnitude in the risks they predict for the low exposure levels of real concern. For a mathematical model to be reliable for extrapolation down to the exposure range of interest, it must be developed on the basis of an adequate mechanistic understanding of the disease process.

For example, to quantify the risk of neurotoxic effects from acrylamide exposure, a number of critical mechanistic factors must be considered. Exposure to high levels of acrylamide is associated with a "dying back" neuropathy in long neurons, the nervous system cells that connect the spine to the arms and legs. A meter or more may separate the long neuron's nucleus (where all protein synthesis occurs) at the spinal end from its synapse (where signals are passed to adjacent cells) at the peripheral end. Connecting the two ends of each cell is the axon, a long channel through which electrical signals are transmitted and various essential biochemical materials are transported. An active transport mechanism moves materials efficiently from their production site in the nucleus to the other intraneuron sites at which they are used. A so-called retrograde system returns used materials to the nucleus for recycling; this is the focus of attention in developing a mathematical model of the "dying back" neuropathy produced by acrylamide that is suitable for risk assessment purposes.

Airborne acrylamide concentrations provide an unsatisfactory surrogate for the dose of acrylamide actually delivered to the axon, which is thought to be acrylamide's specific site of toxic action. Deriving a mechanistic expression of the dose-response relationship thus requires modeling the uptake, metabolism, and distribution of acrylamide to the axon. Acrylamide distributes widely in the body, but detoxifying metabolism occurs primarily in the liver. However, at high concentrations, acrylamide inhibits this reaction; therefore, a nonlinear pharmacokinetic model is needed to predict accurately the amount of unmetabolized acrylamide that will accumulate within axons.

Acrylamide is a highly reactive chemical, and so once it is present within the axons, it can interfere with active retrograde transport by several different mechanisms. First, by binding to glycolytic enzymes, it can inhibit the local energy production that provides fuel for active retrograde transport in the axon. Second, acrylamide can bind directly to a specific protein called dynein and disrupt its critical role in active retrograde transport. Third, acrylamide can bind to the weblike microtrabecular structures present within the axon and thereby increase their viscous drag on the retrograde movement of materials through them. The mechanism of action underlying each of these types of interference defines the shape of the dose-response curve for the particular effect and its impact on retrograde transport. Each is likely to be nonlinearly dependent upon the acrylamide concentration within the axon. Simple "curve-fitting" with generic dose-response models cannot adequately reflect the marvelous complexity and intricacy of this system.

Even for a single chemical, as the case of acrylamide demonstrates, multiple biochemical processes may be involved in the generation of a neurotoxic effect. Possible neurotoxic effects are so diverse that many different mechanisms are likely to be involved, and a unique quantitative dose-response model might be required for each such mechanism, which in turn might be applicable to a few chemicals at most. Until we have gathered considerably more data and experience in this area, the determination of appropriate mathematical models for neurotoxic effects will have to be pursued on a case-by-case basis. There is great intellectual appeal to understanding the underlying biological mechanism that produces a toxic effect, and such understanding would permit development of mathematical models that could be used confidently to predict the risks at exposure levels below those that have been studied. However, for each chemical this development will be a time-consuming and expensive process. As a result, detailed investigations of mechanism will be feasible only for a limited number of chemicals that have widespread exposure or have great potential for commercial development. For the rest, a relatively crude qualitative approach—much like that now used by regulators—will most likely have to be sufficient.

It is possible that some neurotoxicants will not have threshold-like dose-response curves, analogous to what is now assumed to be the case for genotoxic carcinogens. If the public health threat posed by such substances is significant because of very severe neurotoxic effects or high exposure potential, the NOAEL and safety factor approach will be inadequate, and it will be necessary to describe the dose-response relationship in greater detail to choose wisely among the regulatory options. These instances will provide challenging opportunities for extending the science of neurotoxicology and performing more rational risk assessments for neurotoxic endpoints. ■

ENVIRON Opens Texas Offices

ENVIRON's decision to open offices in Houston and Austin reflects both a commitment to its existing Texas and Gulf Coast clients and a *response to the growing need for expert* technical assistance in this marketplace.

ENVIRON is a leader in dealing with the problems that arise from the potential for chemical exposure in the environment and the workplace. The firm's Texas offices provide expert technical assistance and strategic support in the following areas:

- Remedial Investigations and Design of Remedial Programs
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- Litigation Support
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- Multimedia Exposure Assessments (ground water, surface water, air, indirect exposure pathways)
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ROBERT L. MCCOLLOM, JR.

Principal

Robert L. McCollom joins the firm with thirty years of senior management experience in environmental remediation and consulting engineering. Mr. McCollom will be responsible for the development of ENVIRON's Texas and Gulf Coast operations from the Houston and Austin offices.

He previously served as Gulf Coast Regional Manager with Jones & Neuse and as Managing Principal for the Texas operations of Woodward-Clyde Consultants.

Mr. McCollom received his M.S. in Geology from Stanford University and his B.A. in Geology from Dartmouth College.

Robert L. McCollom, Jr.

MICHAEL P. SCOTT

Principal

Michael P. Scott, one of ENVIRON's senior principals, is providing assistance in the development of the firm's Texas and Gulf Coast operations. He brings eight years of experience with ENVIRON, including three years of focused involvement in Texas-based projects.

Mr. Scott received his M.A. in Chemistry from Oxford University and his M.Sc. in Public Health Engineering from the University of Newcastle-Upon-Tyne (UK).

Michael P. Scott

Senior Staff Appointments

■ **Houshang Dezfulian, Ph.D., P.E.**, has joined ENVIRON's Irvine office as Principal. With more than 20 years of experience in geotechnical and environmental engineering, emphasizing the assessment and remediation of hazardous and industrial waste sites, Dr. Dezfulian will strengthen the firm's ability to respond to clients seeking remedial design and engineering. Dr. Dezfulian's expertise in civil and geotechnical engineering, his understanding of the risks of environmental impairment at industrial and hazardous waste sites, and his experience in designing and constructing cost-effective remedial measures will complement ENVIRON's strong chemical exposure and health risk expertise.

Formerly Vice President at Woodward-Clyde consultants, Dr. Dezfulian is a Registered Civil Engineer, Geotechnical Engineer, and Environmental Assessor all in the state of California. He received both his Ph.D. and M.S. in Civil Engineering from the University of California at Berkeley and his B.E. in Civil Engineering from the American University of Beirut in Lebanon. He is the published author of over two dozen scientific and technical papers.

Houshang Dezfulian, Ph.D., P.E.

■ **Edward Hoylman, R.G.**, has joined ENVIRON's *Emeryville office as Principal. Formerly Director of the* Environmental Services Division at Herzog Associates, he has overseen and directed a wide range of environmental projects, many for the solid waste industry. Mr. Hoylman will strengthen ENVIRON's capabilities to provide technical support services for the solid waste industry, underground storage tanks, and water resource development and protection projects.

Within the last four years, Mr. Hoylman has worked at 33 landfills in 13 counties throughout California. This work included siting, geotechnical, closure/postclosure, environmental, and permit assistance for Class *I, II,* and *III* disposal facilities. Mr. Hoylman was responsible for the design and installation of vadose monitoring systems at some of the largest landfills in California. He has worked successfully with federal, state, and county agencies, as well as private disposal facilities.

A Registered Geologist in the states of California and Oregon, Mr. Hoylman received his M.S. in Geology from the University of California at Los Angeles and his B.S. in Geology from the University of Hawaii in Honolulu. He is a Registered Professional Hydrogeologist with the American Institute of Hydrology, and a member of the Society of Exploration Geophysicists.

Edward Hoylman, R.G.

■ **James F. Howard, Ph.D.**, is a new Principal in ENVIRON's Irvine office. He has over 26 years of experience in hydrology, geology, hazardous waste management, ground water development and management, and aquifer remediation. Formerly Principal Hydrogeologist at Harding Lawson Associates, Dr. Howard has experience in all phases of the development, protection, and management of ground water resources, and has directed over 200 projects involving aquifer contamination, monitoring programs, and aquifer remediation.

*Dr. Howard has recently served as a specialist in remote-*sensing applications to hydrogeologic flow, particularly utilizing lineament analysis and associated tech*niques. He has also served as* principal investigator applying remote-sensing technology on numerous investigations involving water supply and contaminant movement.

He holds a Ph.D. in Geology from Indiana University, an M.S. in Geology from the University of Houston, and a B.S. in Geology from the University of Dayton.

James F. Howard, Ph.D.

■ **Deborah A. Barsotti, Ph.D., D.A.B.T.**, is a new Manager in ENVIRON's Princeton office. She has over 10 years of experience in toxicology, with an emphasis on clinical, reproductive, and developmental toxicity as related to heavy metals, pesticides, and halogenated aromatic hydrocarbons.

Prior to joining ENVIRON, Dr. Barsotti was Director of the Division of Toxicology at the Agency for Toxic Substances and Disease Registry. Dr. Barsotti has also served as an expert witness and consultant for cases involving Agent Orange, 2,3,7,8-TCDD, and PCBs; toxicity/adverse drug reactions; and solvent and pesticide toxicity.

A Diplomate of the American Board of Toxicology, Dr. Barsotti received her Ph.D. in Pathology from the University of Wisconsin and her B.A. in Biology (Medical Technology) from Humboldt State University, where she also completed graduate courses in Biochemistry and Electron Microscopy. Dr. Barsotti also currently serves as Adjunct Associate Professor of Toxicology at Drexel University.

■ **David Collins, M.S.**, a new Manager in the Arlington office, joins ENVIRON with over seven years of consulting experience for environmental and engineering projects, including soil and ground water contamination studies; characterization of ground water flow systems; due diligence environmental assessments; and ground water monitoring and production well installation.

Formerly Senior Project Geologist and Project Manager at Environmental Resources Management, Inc., Mr. Collins has managed numerous site investigations and due diligence site assessments. He has also conducted numerous geologic and hydrogeologic investigations with emphasis on ground water sampling programs as a Project Geologist at Walter B. Satterthwaite Associates, Inc.

Mr. Collins is a Certified Professional Geologist in the state of Virginia and a Registered Professional Geologist in the state of Delaware. He has an M.S. in Geological Sciences from Lehigh University and a B.S. in Geology from James Madison University.

Joseph H. Highland, Ph.D., has been named Chief Executive Officer of ENVIRON Corporation. **Robert M. Wenger, Esq.**, has been named President of the firm. **Grover C. Wrenn**, former Chief Executive Officer, has assumed the role of Chief Operating Officer of Applied Bioscience International Inc., of which ENVIRON Corporation is a wholly owned subsidiary.

■ **Randy A. Hagen, M.S., R.G.**, a new Manager in ENVIRON's Emeryville office, has over eight years of experience in performing and supervising hydrologic and soil investigations. He has managed remedial investigations of various types of soil and ground water contamination, including managing Solid Waste Quality Assessment (SWAT) programs for landfills throughout California.

Prior to joining ENVIRON, Mr. Hagen was Project Manager/Hydrogeologist at Herzog Associates, where his work included conducting extensive, multiphase hydrogeologic investigations/characterizations of various California landfills.

A Registered Geologist in the state of California, Mr. Hagen received his M.S. in Geology from the University of Oregon at Eugene and his B.S. in Geology from San Diego State University. He has completed the OSHA 40-hour course in Hazardous Waste Activities Training and is certified in Radiation Safety and Hazardous Materials Waste Handling.

■ **Susan H. Youngren, M.S.**, returns to ENVIRON as a Manager in the Arlington office after two years as a Program Manager at the Risk Science Institute (RSI) of the International Life Sciences Institute (ILSI), where her work included organizing an international conference on the differences between children and adults and the associated implications for risk assessment.

With extensive experience in the areas of exposure assessment, environmental toxicology, and infectious diseases, Ms. Youngren's previous work at ENVIRON included conducting exposure characterizations for Superfund risk assessments; analyzing EPA's method of estimating dietary exposure to pesticides; and developing and coordinating an EPA conference to inform groups affected by EPA actions about their use of risk assessments.

Currently pursuing a Ph.D. in Environmental Biology/Public Policy at George Mason University, Ms. Youngren received an M.S. in Environmental Sciences an Engineering from Virginia Polytechnic Institute and a B.S. in Microbiology and Public Health from Michigan State University.

Publications from ENVIRON

1 **"Generally Recognized as Safe (GRAS) Evaluation of 4-Hexylresorcinol for Use as a Processing Aid for Prevention of Melanosis in Shrimp,"** by V.H. Frankos, D.F. Schmitt, L.C. Haws, et al., in *Regulatory Toxicology and Pharmacology* 14:202-212, 1991.

2 **"Ethylene Thiourea and Secondary Mechanisms in Carcinogenesis: Breaking Out of the No-Threshold Paradigm,"** by B.A. Jackson, L.C. Haws, R.N. Wixtrom, and T.B. Starr, presented at the American College of Toxicology Annual Meeting, Savannah, Georgia, 1991.

3 **"Updating Developmental Toxicity Testing Guidelines for the Safety Assessment of Direct Food Additives and Color Additives Used in Food: Results of a Survey,"** by T.F.X. Collins, T.N. Black, S.L. Graham, B.A. Jackson, and J.J. Welsh, in *Journal of the American College of Toxicology* 10:461-475, 1991.

4 **"Painters Solvent Syndrome,"** by M.A. Katchen, in *Proceedings of the Steel Structures Painting Council Conference*, Long Beach, California, November 1991.

5 **"Ground Water Extraction Systems to Control Both Vertical and Horizontal Migration of Contaminants,"** by R.H. Page, G.A. Cederberg, and M.F. Mohsen, in *Hazardous Materials Control*, Sept./Oct. 1991.

6 **"Effects of Electrol Acceptor on Halogenated Organic Compound Biotransformations in a Biofilm Column,"** by G. Cobb, in *Environmental Science and Technology*, June 1991.

7 **"Evaluation of External Vapor Monitoring Devices for Underground Petroleum Products Storage Tanks,"** by G.B. Wickramayake, R.E. Hinchee, J.A. Kittel, N.G. Reichenbach, and B.J. Nielsen, in *Hazardous Materials Control*, Sept./Oct. 1991.

8 **"Free Petroleum Hydrocarbon Volume Estimates from Monitoring Well Data,"** by G.B. Wickramanayake, N. Gupta, R.E. Hinchee, and B.G. Nielsen, in *Journal of Environmental Engineering* 117(5):686-691, 1991.

9 **"Ammonia Removal from High-Strength Wastewaters by Steam Stripping,"** by G.B. Wickramayake, in *Proceedings of the Hazardous Material and Environmental Management Conference*, 1991.

10 **"Kinetics of the Inactivation of Microorganisms,"** by G.B. Wickramayake and O.J. Sproul, in *Disinfection, Sterilization, and Preservation*, 4th edition (ed. S.S. Block), 1991.

11 **"Disinfection and Sterilization by Ozone,"** by G.B. Wickramayake, in *Disinfection, Sterilization, and Preservation*, 4th edition (ed. S.S. Block), 1991.

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  INDUSTRIAL WASTE COMMITTEE, WATER POLLUTION CONTROL FEDERATION, 1991-93.
  INDUSTRIAL WASTE PRE-TREATMENT TASK FORCE, WATER POLLUTION CONTROL FEDERATION, AD HOC.
- JOSEPH V. RODRICKS, COMMITTEE ON RISK ASSESSMENT OF HAZARDOUS AIR POLLUTANTS, NATIONAL ACADEMY OF SCIENCES (MANDATED BY THE CLEAN AIR ACT OF 1990), 1991-93.

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Exhibit D

Superfund: A program without priorities

By Curtis C. Travis and Carolyn B. Doty

The cleanup of hazardous-waste sites in the United States has become a major industry. The number of sites on the National Priority List (NPL) has reached 1175, and cleanup cost for these sites may reach $30 billion *(1)*. Another $55–$74 billion will be needed for new sites being added to the NPL *(2)*. In an attempt to obtain an overview of the Superfund remedial action decision process, we performed an in-depth analysis of 50 of the 74 decisions signed during fiscal year 1987 *(3, 4)*. We found a large gap between expressed expectations for the Superfund program and reality.

There is a general belief that many people are being exposed to elevated health risks at Superfund sites. The facts do not appear to support this viewpoint. The Agency for Toxic Substances and Disease Registry reviewed 288 NPL sites and found 8% with actual or potential current exposure. Our review confirms this finding.

The most startling statistic we found was that before remediation, 70% of all the Superfund sites had risk levels in the 10^{-4} to 10^{-7} range, the same range that EPA targets as acceptable *after* remediation. Although estimates of future risks were often high, these estimates were based on hypothetical exposure scenarios. We acknowledge that there is an intense public demand for cleanup of hazardous-waste sites. However, given the limited resources of Superfund, the immediate focus should be on identification and cleanup of sites where risk is real and current.

Risk assessment, the cornerstone of EPA's current decision-making process, plays a limited role in defining cleanup priorities. Eighty-eight percent of all sites reviewed were remediated, with little correlation between risk levels and decisions to remediate. All sites with contaminated soils remaining on-site were remediated, regardless of risk levels or the likelihood of migration to groundwater. Risk ranges for contaminated groundwater were essentially identical for sites that were remediated and those that were not. Remediation decisions appear to be driven more by cost, EPA policy, compliance with state and federal environmental regulations, and professional judgment than by current or future risk levels.

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Risk reduction plays a limited role in the selection of remedial alternatives at Superfund sites. Despite the fact that the Superfund Amendments and Reauthorization Act (SARA) mandates a cost-effectiveness approach to the selection of remedial alternatives, the degree of risk reduction associated with remedial alternatives was evaluated quantitatively in only 12% of the decisions. Cost–risk trade-offs cannot be made effectively unless risk reduction is adequately assessed. Therefore, the lack of assessment of risk reduction is a major weakness in the present decision-making process. If protection of public health is to be the primary goal of remediation, assessment of risk reduction must be made an integral part of the remedial alternative selection process.

Ecological risk is not a driving factor in the current decision-making process. Although ecological risks were present at the majority of sites reviewed, no quantitative assessments of ecological risk were conducted and no decisions to remediate were made with reduction of ecological risk as the primary goal. Cleanup levels were set to ensure protection of public health, but with no assurance of protection of surrounding ecosystems. The role of ecological risk needs to be more clearly defined, and environmental assessment methodologies need to be established if assessing ecological risk is to remain a goal of Superfund.

The selection of effective remedial alternatives is essential to the remediation process. However, remedial action decisions are currently made under a cloud of uncertainty regarding effectiveness. Sixty-eight percent of the final remedies reviewed required additional studies to confirm the extent of contamination, the effectiveness of the technology, or its applicability under site conditions *(3)*. Thus, despite the estimated $30 billion cost of remediating sites currently on the NPL, remediation is not guaranteed at the majority of sites.

At some sites, the need for immediate action outweighs the necessity of resolving all uncertainties associated with a remedial alternative prior to selection. However, the majority of sites do not warrant such a degree of urgency. A major need for Superfund is an expanded program to establish the effectiveness of remediation technologies and identify site-specific factors that influence effectiveness.

Another key factor in the attainment of remedial action goals is the selection of permanent remedies. Although SARA emphasizes the selection of permanent remedial alternatives, only 19% of the source remedies we reviewed afford permanence to the maximum extent practicable. Thirty-five

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Exhibit E

14

Problems Associated with the Use of Conservative Assumptions in Exposure and Risk Analysis

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INTRODUCTION

Because many of the qualitative and quantitative factors underlying exposure and risk analyses are uncertain, it is common to choose assumptions, models, and inputs so as to minimize the likelihood that the resulting exposures or risks are understated. The maxim "better safe than sorry" succinctly summarizes the rationale for "conservative," "plausible upper-bound," or "worst-case" approaches to analysis. However, most analyses involve numerous inputs, assumptions, and/or conventions. If conservative choices are made for each, the resulting estimates may grossly exaggerate the actual risks—precluding (or at least undercutting) the development of wise public policy.

The Office of Management and Budget (OMB) in Washington, DC, recognizing that this was (in part) responsible for the less-than-optimal expenditure of some of America's resources, has expressed concern over this issue. Their concern, coupled with Executive Orders 12291 and 12498, certainly suggests that scientists involved in risk assessment make the most reasonable assumptions of exposure, rather than worst-case assumptions, in developing assessments.

This chapter is intended to provide useful guidance for the development of more realistic risk analyses. Seven specific ideas are offered which should improve the quality of risk and exposure analyses.

Most of the specific illustrations in this chapter deal with the polychlorinated biphenyls (PCBs). But our experience suggests that this class of chemicals is not atypical, so the conclusions are more broadly applicable.

The concerns regarding PCBs are due in part to their widespread use, particularly in the 1960s and 1970s, their tendency to bioaccumulate (1, 2), and studies indicating a carcinogenic potential in laboratory animals. In addition, numerous industrial and commercial sites are contaminated with these chemicals. Federal, state, and local regulatory agencies have been struggling to develop realistic policies for controlling human exposure to PCBs, so there is a large data base for examination and review. Several

of these risk assessments are critically evaluated here. A critical analysis of numerous assumptions which can dramatically affect risk estimates is offered. Alternative assumptions and approaches are suggested.

THE SOURCES OF CONSERVATISM: UNCERTAINTY AND COMPLEXITY

The word *conservatism* is used here to mean the selection of assumptions, parameter estimates, models, or procedures that are designed to ensure that resulting estimates of health risks are unlikely to be understated. The wisdom of conservative choices is addressed later in this chapter, but it is first important to understand the sources of conservatism in risk and exposure analyses.

Basically, conservatism is often introduced because exposure and toxicity information are lacking and/or ambiguous. Consequently, there is often considerable uncertainty surrounding key assumptions that enter a risk analysis. For example, in the extrapolation of dose–response data from animal studies to estimate human health risks, sources of uncertainty include:

1. The selection of biological endpoint (e.g., liver tumor, lung neoplasia, total malignancies) for analysis.
2. The appropriate species-to-human dose equivalence or scaling factor (e.g., mg/kg·day, ppm in diet, surface area).
3. The relevant animal data set for analysis.
4. The statistical models and estimation procedures used for extrapolation.

Additionally, risk analysis of commercial compounds may be complicated if, as is the case with PCBs, these are mixtures of compounds rather than "pure" compounds. (Even with "pure" compounds, trace impurities may significantly affect the toxicity.)

Polychlorinated biphenyl, for example, is an operational term given to a series of chemical compounds produced industrially by chlorination of biphenyl with anhydrous chlorine and iron filings or ferric chloride as a catalyst (3). Commercially available PCB preparations contain chlorinated biphenyls with varying degrees of chlorine substitution on the biphenyl ring with the generic formula, $C_{12}H_{10-r}Cl_r$, where r is the total number of chlorine atoms per molecule. PCBs were manufactured in the United States by Monsanto under the trade name Aroclor, and by manufacturers in other countries under other trade names, including Kanechlor in Japan, and Clophen in West Germany. Table 1 shows the approximate percent composition according to degree of chlorine substitution of these commercial mixtures.

The situation is made yet more complex with PCBs than implied by Table 1, because numerous isomers of each of the chlorinated biphenyls are theoretically possible, some 209 congeners in all, and until recently congener-specific analyses were not available. The variability of composition of compounds included in the blanket term *PCB* complicates the preparation of a risk analysis because, as noted below, the various mixtures and individual congeners have different toxicities. Moreover, differing congener-specific environmental transport rates alter the composition of PCBs in environmental media.

In this chapter, the word *uncertainty* is used in a manner consistent with that employed

TABLE 1. Approximate Percentage Composition of Some Commercial PCB Products

	Aroclor Type or Grade							Kanechlors			Clophens	
Chlorobiphenyl	1016	1221	1232	1242	1248	1254	1260	KC-300	KC-400	KC-500	A 30	A 60
$C_{12}H_{10}$	<0.1	11	6	<0.1		<0.1					—	—
$C_{12}H_9Cl$	1	51	26	1		<0.1					1	—
$C_{12}H_8Cl_2$	20	32	29	16	2	0.5		17	3		21	1
$C_{12}H_7Cl_3$	57	4	24	49	18	1		60	33	5	57	2
$C_{12}H_6Cl_4$	21	2	15	25	40	21		23	44	27	17	3
$C_{12}H_5Cl_5$	1	0.5	0.5	8	36	48	12	0.6	16	55	2	20
$C_{12}H_4Cl_6$	<0.1			1	4	23	38		5	13	—	43
$C_{12}H_3Cl_7$				<0.1		6	41				—	25
$C_{12}H_2Cl_8$							8				—	5
$C_{12}H_1Cl_9$							1				—	—
$C_{12}Cl_{10}$											—	—
Average % chlorine	42%	21%	32%	42%	48%	54%	60%	42%	48%	54%	30%	60%

Sources: Polychlorinated Biphenyls, The National Research Council, National Academy of Sciences, Washington, DC, 1979; O. Hutzinger, S. Safe, and V. Zitko, *The Chemistry of PCBs*, CRC Press, Boca Raton, FL, 1980, p. 8; D. N. Paul Michael, Monsanto, personal communication; and Schaeffer et al., op cit. The figures cited for the Clophens are taken from Schaeffer.

TABLE 2. PCB Potency—Lifetime Incremental Health Effects Associated with a Lifetime Consumption of 1 gram of PCB

Risk Probability Calculation	Model	Basis for Animal to Human Conversion	Uses Upper Confidence Bound	Aroclor	Bioassay Data	Calculation
Less than 10^{-10}	Probit	ppm	No	1254	NCI liver carcinoma, adenomas, and hematopoietic system	Maxim and Harrington
3.4×10^{-8}	Logistic	μg/kg	No	1254	NCI liver carcinoma and adenomas	Maxim and Harrington
2.3×10^{-7}	Logistic	ppm	No	1254	NCI liver carcinoma and adenomas	Maxim and Harrington
3.6×10^{-7}	Extreme value	ppm	No	1254	NCI liver carcinoma and adenomas	Maxim and Harrington
3.2×10^{-6}	Logistic	ppm	No	1254	NCI hematopoietic system	Maxim and Harrington
8.7×10^{-6}	Multistage	ppm	No	1254	NCI liver carcinoma and adenomas	Maxim and Harrington
1.5×10^{-5}	One-hit	mg/kg·day	No	1260	Kimbrough hepatocellular carcinoma	OTA (Crump)
1.5×10^{-5}	One-hit	Unstated	No	1260	Kimbrough hepatocellular carcinoma	Decision Focus, Inc., based on EPA-OTS
3.2×10^{-5}	Multistage	ppm	No	1254	NCI hematopoietic system	Maxim and Harrington
3.6×10^{-5}	One-hit	ppm	No	1260	Kimbrough hepatocellular carcinoma	OTA (Crump)
4.2×10^{-5}	One-hit	ppm	99%	1254	NCI liver carcinoma and adenoma	FDA
4.2×10^{-5}	Multistage	mg/kg·day	No	1254	NCI *total malignancies*	EPA–OTS
6.0×10^{-5}	One-hit	ppm	99%	1260	Kimbrough liver carcinoma	FDA
6.8×10^{-5}	Multistage	mg/kg·day	95%	1254	NCI total malignancies	EPA–OTS
1.9×10^{-4}	One-hit	ppm	99%	1254	NCI total malignancies	FDA
2.5×10^{-4}	Multistage	$\mu g/(kg)^{2/3}$	No	1254	NCI total malignancies	EPA–OTS
3.2×10^{-4}	Multistage	mg/kg·day	No	1260	Kimbrough hepatocellular carcinomas and neoplastic nodules	CAL–Health, recomputed
4.1×10^{-4}	Multistage	$\mu g/(kg)^{2/3}$	95%	1254	NCI *total malignancies*	EPA–OTS
1.9×10^{-3}	?	Surface area	No	?	?	CAL–Health
2.2×10^{-3}	?	Surface area	?	1260	Kimbrough	EPA–OHEA
2.7×10^{-3}	?	Surface area	95%	?	?	CAL–Health
4.3×10^{-3}	Multistage	Surface area	95%	1260	Norback and Weltman	EPA cited in ATSDR

by Whipple (4): "a lack of definite knowledge, a lack of sureness; *doubt* is its closest synonym" (see also Wilson and Crouch, 5). Risk, in contrast, is used to measure the probability and severity of loss or injury. Both risk and uncertainty may be couched in probabilistic terms; the lack of predictability arises from insufficient knowledge in the case of uncertainty rather than from the probabilistic outcome of a well-understood stochastic process in the case of risk.

Real uncertainties often surround the analysis of dose–response data (points 1–4 above) in the case of most chemicals, including PCBs. The selection of the appropriate assumptions and models is often ultimately judgmental. Even the interpretation of the basic data is seldom unequivocal. Barnard (6), for example, illustrates data interpretation problems for bioassays with the case of nitrites:

> Problems of evaluation arise not only because of the mass of experimental data and questions of their validity, but also because the interpretation of the data is ultimately judgmental. A well-known example is nitrites. The original bioassay was reported to be positive; great public uproar and regulatory activity took place because nitrites are important antibacterial additives to meat. Upon full peer review of the data, the study was judged to be negative.

Dose–response model identification and estimation involve substantial uncertainty (7–11), as does the choice of scaling factors [e.g., on a weight versus a surface area basis, (12)], and the other elements above. Depending on how these uncertainties are handled, the estimates of human health risks for a chemical under investigation can differ by orders of magnitude. Table 2, for example, shows a sampling of potency values—here shown in approximate terms as the lifetime incremental health risk associated with the consumption of 1 g of PCBs—taken from the literature. For example, the 1.9×10^{-4} risk per gram estimate in Table 2 is derived as follows. Cordle, Locke, and Springer (13) estimated that 50th percentile fish eaters consumed an average of 8.46 μg PCB/day. The corresponding lifetime dose is 8.46×10^{-6} g/day times 365 days/yr times 70 yr/lifetime, or 2.16×10^{-1} g PCB/lifetime. Based on National Cancer Institute (NCI) data, Cordle et al. concluded that eaters of contaminated fish would experience an incremental lifetime health risk of 4.1 per 1000,000. Thus, the incremental lifetime risk associated with the consumption of 1 g of PCB is $4.1 \times 10^{-5}/2.16 \times 10^{-1}$ or 19×10^{-4}. This estimate used total malignancies, from the NCI study as the endpoint, a 99% statistical upper confidence estimate of the dose–response slope of a one-hit model, and based the species-to-human conversion on the assumption that equal parts per million (ppm) in the diet would produce an identical response.

The risk estimates given in Table 2 span approximately seven orders of magnitude from less than 10^{-10} to 4.3×10^{-3}. Although it may be argued that not all the estimates in Table 2 are equally likely, all have some degree of scientific credibility. Similarly broad ranges in risk have been observed for vinyl chloride, saccharin, and other compounds (14), so the estimates for PCBs should not be viewed as atypical. What then is a "reasonable" way to deal with this uncertainty? (We are mindful of Ambrose Bierce's cynical definition of reason: "to weigh probabilities in the scales of desire.")

Although numerous approaches have been proposed for quantifying and otherwise treating uncertainty, regulatory agencies have generally opted to choose the most conservative among the alternative risk estimates. For PCBs, this means (expressed in risk/gram terms) accepting a potency of the order of 10^{-3}, rather than any smaller estimate. For example, the recent Agency for Toxic Substances and Disease Registry (ATSDR) draft Toxicological Profile for PCBs (15) cites a potency (expressed in the units

given in Table 2) of 4.3×10^{-3} without any mention of the fact that this estimate is the largest among many alternatives.

As noted, one of the issues that serves to complicate the selection of a potency estimate for PCBs relates to the variable composition among the commercial mixtures coupled with evidence that the toxicity varies among these mixtures. For example, an NCI study on Aroclor 1254 stated (16):

> It is concluded that under the conditons of this bioassay, Aroclor 1254 was not carcinogenic in Fischer 344 rats.

But the study generally regarded as providing the most convincing evidence of the carcinogenicity of PCBs (17) is that conducted by Kimbrough et al. (18). This Kimbrough study used Aroclor 1260, a mixture containing approximately 60% chlorine (q.v., Table 1). Moreover, the Norback and Weltman study (19) (used as the basis for EPA's recent potency estimate) also used Aroclor 1260 (of unreported purity) for its feeding studies. That these latter studies used only Aroclor 1260 is important because there is strong evidence from numerous studies that the biological activity of PCBs is a function of the degree of chlorination:

1. Feeding experiments over 224 days with Kanechlor 300, 400, and 500 in mice were conducted by Ito et al. (20). Hepatocellular carcinomas were induced only by the highest chlorinated compound, Kanechlor 500 (q.v., Table 1).
2. A study by Schaeffer et al. (21) indicated that at the end of an 800-day feeding experiment, the incidence of hepatocellular carcinoma in rats fed Clophen A 60 (similar to Aroclor 1260 q.v., Table 1) reached 48%, whereas only 3% of those fed Clophen A 30 and 0.8% of the controls were similarly affected. It is unfortunate that ATSDR remarked (p. 72) only that the Schaeffer study "demonstrates that PCB mixtures free from contamination with furans elicit a carcinogenic response." (The study reported that the Clophens were free of chlorinated dibenzofurans, but test method, level of detection, and actual results were not specified.) In fact, the Schaeffer study has much broader implications. Table 3 shows the Schaeffer data. The incidence of hepatocellular carcinoma was elevated (in a statistically significant manner) only for Clophen A 60. Thus, the results of this study not only supported other findings that PCB mixtures containing 60% chlorine by weight were associated with hepatocellular carcinoma in rats, but also indicated that PCBs containing lesser amounts of chlorine were not

TABLE 3. Frequency of Hepatocellular Alterations Induced by Chronic Feeding Studies with Clophen A 30 and Clophen A 60

	Number of Foci	Neoplastic Nodules	Hepatocellular Carcinoma
Controls	6/131	5/131	1/131
(group 1)	(4.5%)	(3.8%)	(0.8%)
Clophen A 30	63/130*	38/130*	4/130
(group 2)	(48%)	(29%)	(3%)
Clophen A 60	3/126	63/126*	61/126*
(group 3)	(2.4%)	(50%)	(48%)

Source: Schaeffer et al., as cited in Harbison et al.

*Denotes a significant difference from the control value ($P = 0.05$).

proven to be carcinogenic. Even if it is argued that Clophen A lack of significance was solely an artifact of sample size, the raw data is consistent with the finding that the potency of A 30 is at most 1/16 that of A 60! This finding is absolutely at variance with the assumption made by EPA and ATSDR that "all PCBs have equal potency."

3. Schaeffer et al. (21) also note,

> Both the DHEW Subcommittee on Health Effects of PCBs and PBBs (1978) and Ecobichon (1975) have reported that the toxic potency of PCBs (hepatic enzyme induction, hepatocarcinogenic effect) *increases with increasing chlorination and chlorine substitution in the para, ortho,* meta positions, respectively.

These and other results support the notion of increasing biological hazard with increasing average degree of chlorination of PCB mixtures. Thus, Kimbrough's and Norback and Weltman's results with Aroclor 1260 have to be viewed as a "worst case," and moreover, an "unlikely worst case" as production of 1260 only accounted for a minority of total domestic PCB production (21). (According to Monsanto, production of Aroclor 1260 accounted for only 10.6% of total Aroclor production over the period 1957–1977.)

Uncertainty is not confined to potency estimates. Some of the quantities used in a risk-exposure analysis include the following:

1. Initial contaminant concentrations.
2. Physicochemical constants to describe the kinetics of contaminant transport between environmental compartments.
3. Exposure frequency of humans to contaminants in various environmental compartments.
4. Human contact (uptake) rates (e.g., soil, accretion rates, soil ingestion rates, respiration rates, consumption rates for various foodstuffs) for alternative exposure pathways.
5. Bioavailability fractions (e.g., absorption rates through skin).
6. Dose–response parameters and models.

Many of the parameters, constants, and variables in models relevant to 1–6 are not known with accuracy. As simple a quantity as soil ingestion rates—a key parameter in many PCB risk analyses—for example, admits to numerous estimates, such as are shown in Table 4. These estimates range over almost three orders of magnitude, from 25 to 10,000 mg/day.

Faced with these uncertainties and complexities, the regulatory response has generally been to avoid making choices that could potentially lead to underestimates of human health risk as illustrated above with potency estimates for PCBs. In some cases, this response has been implicit. But in others, conservative assumptions have been explicitly mandated by regulatory agency policy. For example, the carcinogen assessment guidelines employed by numerous federal and state regulatory agencies are clearly conservative, a fact acknowledged in a recent critical report by the Executive Office of the President, Office of Management and Budget (38), as shown in Table 5.

The conceptual rationale for conservatism in exposure and risk analysis is a perception among regulatory agencies that the "social cost" of "false positives" (incorrectly judging a contaminant to present a hazard) is less than that of "false negatives," a theme developed at length in a thoughtful article by Whipple (4).

TABLE 4. Estimates of Daily Soil Ingestion from Various Sources

Age	Daily Ingestion Rate (mg/day)	Source	Remarks
Toddlers	100	Barltrop (22) 1973	
?	33	Bryce-Smith 1974	Cited in Duggan and Williams (23)
4.3 years (mean)	100	Lepow et al. (24, 25) *1974, 1975*	Based on observations of mouthing behavior
1–3 years	10–1,000	Day et al. (26) 1975	Based on ingestion of soil on candy
2–6 years	25	Duggan and Williams (23) 1977	Estimate of amount of street dust that urban children ingest daily
1–3 years	140–430	Mahaffey (27) 1977	Based on estimate of paint consumption by children with pica
Various	40	National Research Council (28) 1980	Recommended for the sake of exposure calculations
Various unspecified	100	EPA (29, cited in 28) 1983	Lead in air study
2–6 years	100–5,000	Schaum (30) 1984	Upper end of range represents habitual *pica consumption*
0–9 months	0	Kimbrough et al. (31)	Reportedly based on study of lead exposure; generally thought to be an overestimate
9–18 months	1,000		
1.5–3.5 years	10,000		
3.5–5 years	1,000		
5 years	100		
2.5 years	164	Hawley (32) 1985	Includes both soil and dust ingestion; rates calculated on 365-day/yr basis
6 years	24		
Adults	61		
1–3 years	180	Binder et al. (33) 1985	Based on measurement of trace elements—upper 95% percentile less than 600 mg/day
0–3 years	2,500	Dime (34) 1985	Used in state of New Jersey risk analysis for PCBs
Various	100	EPA (35) 1985	Superfund health assessment manual
0–1 year	50	Clement Associates (36) 1986	Used in Smuggler Mountain endangerment assessment; meant to summarize literature estimates
1–6 years	100		
6–11 years	50		
Over 11 years	20–50		
1.5–3.5 years	100	Paustenbach et al. (37) 1986	Based on complete review of literature

TABLE 5. OMB Characterization of Cancer Assessment Models Emplo_ by EPA and Other Federal Agencies

A few examples of these cautious or conservative assumptions are: (1) treating all benign tumors as malignant, (2) using data about only the most sensitive animal species and sex, and (3) using conservative mathematical models to extrapolate from high to low doses. Each of these three kinds of assumptions is discussed briefly below.

All benign tumors treated as malignant. In interpreting animal studies, agencies frequently interpret both benign (noncancerous) tumors and malignant (cancerous) tumors to be equally strong indications that a substance is a carcinogen. Scientists know, however, that not all benign tumors evolve into malignancies. Studies that treat benign tumors the same as malignant tumors can overstate the real risk present. Some risk assessments based on animal studies have concluded that a chemical is carcinogenic solely because of an increased number of benign tumors. Assuming that all benign tumors will become malignant will not produce a best estimate of the risk.

Use of most sensitive species and sex. Even though the results of several animal studies may be available for a particular suspected carcinogen, it is not unusual for the risk estimate to be derived only from the data for the most sensitive exposed species and sex. This conservative approach tends to overpredict the risk to humans, because it assumes that humans are as sensitive as the most sensitive animal tested even when the most sensitive animal tested is hundreds of times more sensitive than any other animal tested. Furthermore, by using the same data to derive the risk estimate and to determine the most sensitive species, the chance is increased that statistical anomalies will lead to overestimates of the risk. (If a statistical anomaly causes an upward bias in the estimated risk for a particular species, it will also increase the chance that that species will be selected as the most sensitive.) A more accurate estimate could be derived from a weighted average of all the scientifically valid, available information.

Conservative extrapolation from high doses to low doses. To determine the risks to humans from exposure to a substance, scientists must extrapolate (or estimate) from the results of high doses in animal experiments to the comparatively low doses of human exposure. This extrapolation relies upon statistical models. The risk from exposure to low doses cannot be determined with certainty. In making the extrapolation, the common practice is not to make a best estimate of the risk from human exposure to low doses, but to determine what a maximum risk would be. Often, such an extrapolation has a 95 percent chance of overstating the true risk. Usually, the explanation for using these conservative assumptions is to ensure that the actual risk is not underestimated. However, the resulting risk estimate can be over one hundred times greater than the best estimate of the risk.

Whipple suggests another factor that encourages conservatism (39):

> An additional factor encouraging conservatism is how a regulatory agency's decisions might be judged in hindsight. An overcontrolled risk will probably drop from sight once a decision is implemented and control investments made, despite continuing social costs. But an undercontrolled risk, possibly discovered through the identification of victims, is far more disturbing for a regulatory agency.

CONSERVATISM CHALLENGED: EVOLVING PERCEPTIONS AND EXECUTIVE ORDERS 12291 AND 12498

Notwithstanding the above, there appears to be a growing awareness in the regulatory community that (i) conservative assumptions can significantly overstate risks, (ii) such overstatement could actually be ultimately counterproductive, and (iii) more realistic risk estimates are appropriate.

Table 6 presents a selection of observations from regulatory personnel, environmentalists, and academics that address uncertainty, conservatism, and the consequences

TABLE 6. Uncertainty, Conservatism, and Resulting Consequences in Risk Analysis

Statement	Reference/Remarks
Historically at EPA it has been thought prudent to make what have been called conservative assumptions; that is, our values lead us, in a situation of unavoidable uncertainty, to couch our conclusions in terms of a plausible upper bound. This means that when we generate a number that expresses the potency of some substance in causing disease, we can state that *it is unlikely that the risk projected is any greater.*	W. D. Ruckelshaus, former EPA Administrator (40)
This is fine when the risks projected are vanishingly small; it's always nice to hear that some chemical is not a national crisis. *But when the risks estimated through such assessments are substantial, so that some action may be in the offing, the stacking of conservative assumptions one on top of another, becomes a problem for the policymaker.* If I am going to propose controls that may have serious economic and social effects, I need to have some idea how much confidence should be placed in the estimates of risk that prompted those controls. I need to know how likely real damage is to occur in the uncontrolled, partially controlled, and fully controlled cases. Only then can I apply the balancing judgments that are the essence of my job. [emphasis added]	
I'm skeptical of quantitative risk assessment, at least in the cancer field. The science is too imperfect, and the results are likely to be used literally, because all the caveats get lost.	K. Ahmed, Research Director for the Natural Resources Defense Council (41)
Milton Russell, Assistant Administrator for Policy, Planning, and Evaluation at EPA, added that "depending on which animal you use, and whether you use a model that uses surface area or weight, you can get a difference in risk of up to 39,000 times." He went on to add that uncertainties in the risk assessment process are multiplied (not added) and in the case of cancer risk this leads to extreme conservatism in the decision-making process. "If you are relatively sure of the probability of risk, like automobile accidents, the range of uncertainty is narrow, and the difference between a plausible upper bound and a maximum likelihood and a plausible lower bound is relatively small. But if you are quite uncertain (as we are in many of these health effects), the range between this upper and lower bound is very, very large. *Multiplying the large uncertainties associated with each factor in the estimate leads to cascading conservatism in decision making.*" [emphasis added]	B. Barker (41)
Often each conservative assumption is made by a different scientist or analyst responsible for a portion of the risk assessment. Each may think that erring on the side of caution or conservatism is reasonable. However, the effect of these individual conservative assumptions is compounded in the final estimate of risk presented to the decisionmaker.	OMB (42); see also Nichols and Zeckhauser (12) for a numerical example

TABLE 6. (*Continued*)

Statement	Reference/Remarks
In practice, there may be as many as 20 distinct stages in a risk assessment where conservative assumptions are made. A typical risk assessment would probably contain about 10. The final risk estimate derived from these compounded conservative assumptions may be more than a million times greater than the best estimate and may, thus, have a probability of being accurate that is virtually zero. Some combinations of these highly cautious assumptions so overstate the risk that they are unrealistic.	
More recently, EPA has adopted the multi-stage model which has a linear component at low doses (4). This model assumes that cancer is caused by a series of mutational steps, whose occurrence [sic] rest both on a dose and potency. This model also results in a conservative estimate. Most scientists accept these models as giving plausible upper limit estimates for a chemical's potency at low levels of exposure. *In other words, the potency of a substance is unlikely to be higher that* [sic] *estimated using the linear model, but could be substantially lower. Use of the linear non-threshold models reflects EPA's decision to err on the side of caution in the face of uncertainties.* The final result of the linearized extrapolation is a "unit-risk factor," which gives the estimated upper limit lifetime risk per unit of exposure. [emphasis added]	D. R. Patrick (43)
These gaps in our scientific understanding and data limitations imply that it is difficult to conduct a good risk assessment. It is no surprise that they vary in quality. The many stages where judgment must be applied make it very easy for the results to substantially overestimate or underestimate the unknown true risks. *Because a government agency's mandate typically is to protect the public, or to be safe rather than sorry, the cumulative effect of these conservative assumptions may be very large. The resulting risk estimates often are treated as plausible upper bounds.* Unless the uncertainty associated with each assumption is stated, risk managers often view these risk estimates as actual risks. [emphasis added]	A. Fisher, EPA (44)
The Agency is not alone in its concern that different assumptions and different mathematical models used can significantly alter the outcome of risk assessment. When the Occupational Safety and Health Administration (OSHA) published its cancer policy in 1980, it did detailed comparisons of how estimates of carcinogenic risk can vary with the assumptions used in developing the estimates (45 FR 5198–5200). By varying the method of low dose extrapolation used, and the toxicology or epidemiology study which formed the basis of the risk assessment commenters to the OSHA policy developed risk estimates for exposure to 1 ppm of vinyl chloride which ranged from 10^{-8} (one in one hundred million) to 10^{-1} (one in ten, or 10%). A similar exercise with saccharin by NAS, and reprinted	United States Environmental Protection Agency (45)

TABLE 6. (*Cont...*)

Statement	Reference/Remarks
in the OSHA policy (45 FR 5200), estimated the expected number of cancer cases in the general population (exposed at 0.12 grams/day) at between 0.001 cases per million exposed, and 5200 cases per million exposed. These differing estimates were developed by using different low-dose extrapolation models and different animal-to-human extrapolation methods—all of which had some credence in the scientific community.	
Recent research has also shown a need to reevaluate the role of "conservatism" in assessing and managing risk. Making a "conservative decision" (i.e., one that is likely to be more protective of health and the environment than an alternative decision) is widely accepted as a prudent practice in risk management. *In keeping with the recommended separation of risk assessment and risk management activities, however, conservative assumptions, conservative models, conservative estimates, etc., should not be key elements in the science-based risk estimation steps. A catenation of conservative assumptions, models, and estimates throughout a risk assessment can lead to a "worst-case" (or even worst-of-the-worst-cases) prediction that may be of little value* (or possibly misleading) *to the decision maker.* Most decisions actually involve "either–or" choices between technological alternatives with different risk levels rather than a "yes–no" choice on a single risk. When dissimilar alternatives require different analysis procedures, conservatism ambiguously or inconsistently applied could lead to biased results and poor decisions—even to the choice of a technology that is less protective of human health and the environment and possibly more costly to society than an available alternative. Best estimates of the risks, costs, and benefits for the alternatives, coupled with consideration of their uncertainties (including worst-credible case considerations), should produce the optimal basis for decision making. The Council on Environmental Quality has recently noted that "rules of reason" should replace worst case analysis as the basis of regulatory decision making. [emphasis added]	Midwest Research Institute (46)

to risk and exposure assessment. It is particularly noteworthy that senior officials at EPA are beginning to understand the problems occasioned by making conservative choices at several points in the analysis and to rethink the wisdom of these procedures.

As indicated by these quotes, current thinking appears to be shifting away from the "better-safe-than-sorry" premise toward the development of models and selection of assumptions that more accurately portray the actual risks. The place for conservatism (if at all) should be in the *risk management* rather than the *risk analysis* phase of regulatory action. Raiffa (47), Chairman of the Committee on Risk and Decision Making, National Research Council, offered the following suggestion:

> *Probabilistic reports should not prejudice policy issues and purposely report with a prudent bias.* Cascading prudent reports could result in imprudent actions, and there is a danger of double-counting competing risks. Such reporting should be honest, and not attempt to second-guess policy choices. Probabilistic reports about diverse consequences to health, for example, are very often slanted to be conservative. I believe that it is better to report honestly, and that *prudence should, more appropriately, be accounted for in the evaluation process, rather than in the assessment process.* [emphasis added]

Barnard (48) echoed these comments in an essay on the partnership between law and science in risk analysis and risk management:

> It is sometimes said that the scientific evaluation of risk should be "conservative" because it deals with human health. But this puts "conservatism" in the wrong place in the regulatory structure. It is the function of the regulator to apply the social criteria of cost, safety, reasonableness, and acceptability. It is in making these decisions that "conservatism" may play a role. If a scientific evaluation is constrained in the name of "conservatism" by social values or management policy, the result will be biased in unobvious ways. Such an evaluation does not provide a sound basis for the difficult social/legal decisions a regulator must make.

Nichols and Zeckhauser (49) also address the problems resulting from blurring the distinctions between risk analysis and risk management:

> In practice, the line between risk assessment and management is often blurred. Fundamental gaps in scientific knowledge and data limitations make risk assessment a highly uncertain endeavor, requiring many choices among competing models and assumptions. There is a strong temptation to have such choices reflect implicit policy judgments rather than science.
>
> This blur is most apparent in current techniques for estimating the risks associated with carcinogens, which employ conservative assumptions that bias the estimates upward. The intent is to err on the side of safety by minimizing the chance that risks will be underestimated and thus undercontrolled. But this approach intrudes the risk assessment process into risk management. In deciding how conservative to make their estimates, risk assessors implicitly trade off risk against other factors. Unless they explicitly acknowledge these trade-offs and quantify them, their assessments will mislead others, including those charged with managing risks. For example, risk managers will be more likely to impose a costly regulation if they mistakenly believe that it can prevent ten cases of cancer than if they correctly realize that it will eliminate only one case.
>
> Advocates of the conservative approach are likely to view this tendency toward greater control as a virtue; conservatism is intended to give extra weight to protecting public health and, under conditions of massive uncertainty, to err on the side of safety. In fact, however, conservative risk assessment is a deeply flawed approach to protecting public health. It violates the distinction between risk assessment and risk management, concealing value judgments and policy choices under a cloak of science. It creates capricious differences in the degrees of safety provided across different substances and policy areas, because degrees of conservatism vary widely. Finally, because regulators must make complex trade-offs among different risks (not only between risk and cost), conservation can lead to less rather than more safety, by misdirecting public concern and scarce agency and societal resources.

Many of the above ideas (including those given in Table 6) are addressed (explicitly or implicitly) in Presidential Executive Orders 12291 (February 17, 1981) and 12498 (January 4, 1985) directed broadly at regulatory reform. Executive Order 12291 requires benefit-cost analysis of major federal regulations. Executive Order 12498 reaffirmed these

guidelines and explicitly addressed health and safety matters directly, stating (50) that "regulations that seek to reduce health or safety risks should be based upon scientific risk assessment procedures, and *should address risks that are real and significant rather than hypothetical or remote*" [emphasis added]. Such language is pointedly directed toward increasing the realism of risk analysis. Indeed, "improving coordination and consistency in risk reduction" was one of the principal themes in the recent Executive Office of the President, Office of Management and Budget (OMB) 1986–1987 Regulatory Program. In particular, this document defines the regulatory agenda for implementation of the above referenced Executive Orders. Improvements to risk assessments were a major topic of this report. OMB was strongly critical of the conservative assumptions often employed in carcinogen risk and exposure assessment (see Table 5) and highlighted the reasons why such practices were problematic (51):

> *Risk Assessments with such extreme conservative biases do not provide decisionmakers with the information they need to formulate an efficient and cost-effective regulatory strategy.* Furthermore, the inconsistency of these assumptions makes it virtually impossible to compare risks from different sources. It is particularly difficult to compare safety risk estimates, which are usually best estimates, with health risk estimates, which usually are not best estimates, because the latter embody a series of conservative assumptions. Even different estimates of health risks may not be comparable because of the different degrees of conservatism built into them. Where risk estimates for two different risks cannot be compared, it will be impossible to compare the effects of regulations controlling them.
>
> *A perverse and unfortunate outcome of using upper-bound estimates based on compounded conservative assumptions is that it may lead us to regulate insignificant risks and ignore more serious risks.* Furthermore, the more uncertain we are about the risk posed by a particular hazard, the higher the upper-bound risk estimate will be. Therefore, the less information we have on the risk posed by a potential hazard, the more likely we are to regulate it. Other hazards that pose certain but smaller risks are not considered as dangerous and may not be regulated. Yet, hazards with better understood risks may be more serious.
>
> *All the problems we have discussed resulting from compounding conservative assumptions can be addressed by developing best estimates at each stage of the risk assessment process.* Estimates of the uncertainty and the outer ranges of potential risk can be developed to supplement the best estimate. Both the best estimate and these supplementary risk indicators should be made available to decisionmakers. Then, if regulatory decisionmakers want to choose a very cautious strategy of risk control, they could do so and a margin of safety could be applied at the final decision and would be based on all the available information about its consequences and those of alternative strategies. The public and affected parties would also benefit from knowing both the expected risk and the margin of safety rather than being given only alarming and inconsistent estimates that are likely to be very different from actual risks.
>
> *Only when best estimates of risks and other information on the likely level of risks are presented to the decisionmaker, rather than hidden in the assumptions, can we be sure that we are issuing regulations that will make society as well off as possible.* Fortunately, more review by regulating departments and agencies and by the Executive Branch has already begun to improve consistency in risk assessment and risk management and, thereby, improve societal welfare. Executive Order No. 12291 provides a mechanism to help ensure consistency. [emphasis added]

The above quotation—and extended discussion from which it was extracted—underscores the desirability of and executive branch emphasis on the need for realism in risk analysis. It remains to identify ways to operationalize this idea. How can risk assessments be made more realistic?

SEVEN SPECIFIC SUGGESTIONS FOR IMPROVED RISK AND EXPOSURE ANALYSES

Shown below are seven suggestions to make risk and exposure assessments more useful to risk managers. The items in this list are neither exhaustive nor mutually exclusive, but experience has proven that the ideas contained are useful.

1 Emphasize Best Estimates Rather Than Extremes

As noted, many of the assumptions and numerical inputs to a risk analysis are not known with certainty. In this circumstance, it is certainly appropriate to consider "conservative" values to bound risk estimates, but it is even more important to attempt to select "best" or "most likely" sets of assumptions for risk assessment. As OMB notes (52), with respect to measures of central tendency:

> This measure is favored by statisticians, economists, and scientists because it is the most accurate measure of what, on average, is likely to happen. The use of the best estimate does not preclude the supplementary use of other measures in order to understand the variation around this average, such as the variability of the risk or its upper or lower bounds. Agencies often—and should—use these supplemental measures as well. Using the best-estimate approach along with estimates of uncertainty allows policymakers to understand the range of possible risks and to choose the margin of safety that is appropriate for specific regulations. A regulatory agency could choose to be very cautious and regulate to protect people against a risk that has a very small chance of actually occurring, i.e., a risk at the higher end of the range of uncertainty. An agency could choose to regulate so that the chances of a risk occurring are, for example, only 5 percent, 1 percent, or 0.01 percent. However, it could be very costly to regulate to this level.
>
> Risk assessments of health hazards—as opposed to safety hazards—particularly those based on animal tests, rarely develop a best estimate of the risk. Instead, such risk assessments of health hazards often inform the regulatory officials and the public of only the high end of the range of *uncertainty of the risk, i.e., only what the most cautious estimates are.* Regulations based upon these so-called upper-bound estimates may, therefore, address a risk that is almost nonexistent. When agencies focus their efforts on regulating insignificant risks, they may end up ignoring other more significant risks.

Table 2, noted earlier, shows the range of potency estimates reported for PCBs and illustrates the present emphasis on extreme rather than most likely values. The values most often employed in risk analyses of PCBs are at or near 10^{-3} in risk/gram units [*equivalent to a potency of approximately* 7.7 $(mg/kg \cdot day)^{-1}$]. A more reasonable estimate is likely to be nearer the neon or median of the estimates given in Table 2, say approximately 10^{-5}, yet few analyses have employed this figure.

Even when mean values are employed in an analysis, other conventions may serve to bias mean values upward, and the analyst must be careful to spot these biases. For example, a recent endangerment assessment (EA) conducted at the Northside Sanitary Landfill (NSL) based some exposure scenarios on the maximum reported contaminant concentration. *Recognizing that these maximum values might overstate exposures,* the analysis also estimated risks associated with mean concentrations. In calculating these mean values, the authors of the report noted (53):

> Several arbitrary conventions were used in calculating means that may bias the means. For nondetectable results, method detection limits were used for calculating the arithmetic means. Using method detection limits may bias the mean high where it is reasonable to believe contaminants were not present; however, this is consistent with current EPA policy.

Taken to its limit, such a procedure is preposterous—every site could have an arbitrarily high cancer risk by simply adding (and calculating risks for) chemicals not detected in the sampling protocol! The NSL EA did not go to this extreme, but resolved the problem by omitting calculation of means when the proportion of "nondetects" exceeded 20%—and reporting only risk estimates based on maximum values for cases where this occurred. In fact, there is a well-developed statistical theory for estimating means from truncated *samples, so no such ad hoc rules are required (54). (In the future, this may be less of a* problem because detection limits are generally decreasing as new analytical procedures are being developed.)

Best estimates are important in exposure calculations. OMB challenged the use of worst-case environmental conditions in exposure calculations (55):

> Use of worst-case environmental conditions. To estimate what concentrations of a contaminant reach a point at which humans might be exposed, a chemical's movement through the air, water, or soil usually must be estimated with a computer model. Movement of a chemical, for example, depends greatly on environmental conditions, such as windspeed and direction for airborne pollutants; surface water flow, acidity, and temperature for water pollutants; and groundwater velocity, flow, and soil type for chemicals that pass through the soil. Often, only one calculation is made for the entire Nation on the assumption that it is impractical to set different regulatory standards for different environmental conditions. When a single model must represent conditions for the whole Nation, agencies frequently assume the unique circumstances that together may present the greatest risk, and then assume that this circumstance exists everywhere in the Nation. For example, a gravel soil environment (rather than clay or some other soil condition) might be used in a model because chemicals in groundwater move most quickly through gravel and, thus, are likely to pose a greater risk. However, since not all soil is gravel, this assumption will overstate actual risks.

Other examples of worst-case reasoning are legion. In an occupational setting, for example, EPA, OSHA, and state agencies often assume that a worker is exposed for as much as an entire working lifetime (from age 20 to age 65) or 45 years. To illustrate, in an assessment of reentry guidelines for PCB- and TCDD-contaminated surfaces at the Binghamton (New York) State Office Building (56), it was assumed that workers would be exposed over a 30-year period. Likewise, a similar analysis by the California Department of Health Services for reentry guidelines for the One Market Plaza building (57) assumed that workers would occupy the building for 40 years. Although the possibility exists that a worker could remain at one job location for as much as a 30- or 40-year period, it cannot be termed a "most likely" value in any realistic sense. National estimates by Hall (58) suggest that only 7% of U.S. workers would hold one job—let alone to be employed at one location—for 35 years or more. The Department of Labor, Bureau of Labor Statistics (BLS), publishes data on "ongoing tenure"—and estimates of "completed tenure" have been made using these data (59). Hall (60), in particular, estimated a median completed tenure of 7.7 years for all U.S. workers in 1978, based on BLS data. Completed tenure estimates will *overstate* the expected additional years of exposure, because the completed tenure figure includes time already spent on the job (ongoing tenure) as well as the *expected additional tenure. The median ongoing tenure of all workers is approximately 3.2* years (61), so to a first approximation, the likely additional tenure is $7.7 - 3.2 = 4.5$ years, or rounded, 5 years. Five years would have been a much more realistic estimate of the likely occupational exposure for an occupant of the Binghamton or One Market Plaza office buildings. Had this and other more realistic assumptions been employed in the risk assessment at these locations, it is possible that quite different cleanup standards would have prevailed.

As a second example, a residential exposure scenario for evaluation of groundwater contamination typically posits that exposed humans ingest 2 liters of water per day—all taken from the same contaminated source—over a 70-year nominal lifetime. Yet actual studies on water consumption support figures closer to 1 liter per day (62), which includes the contained water in carbonated beverages (unlikely to contain the contaminant) and prepared hot drinks such as coffee (where heating might partially evaporate or otherwise remove or destroy contaminants). And, in any event, the assumption of 24-hour per day occupancy over a 70-year period cannot be termed plausible.

Critics might well argue with the above examples and point out that building occupants or residents who depart will be replaced by others and that, in any event, it matters little to whom the risk occurs. Even if this premise is accepted, and a person-years metric is substantial for the calculation of individual risks, the above examples cannot be dismissed lightly, particularly when considering contaminant depletion. Succeeding generations of occupants will force lower exposure levels if the contaminants decay over time as discussed below.

At least some elements within EPA are sympathetic to the need to develop more realistic exposure scenarios. For example, EPA is reportedly considering substituting a residential exposure scenario of 16 hours per day for 10 to 35 years rather than 24 hours per day for 70 years (63).

A point often overlooked in exposure analyses is pollutant depletion/removal. Over time, pollutants are depleted by a variety of mechanisms, including volatilization, erosion, dilution/dispersion, photolysis, chemical reaction, and biodegradation. These transport processes reduce the contaminant concentration and hence potential for exposure. Although some exposure and risk analyses have explicitly treated depletion (e.g., 64–66), others have omitted this phenomenon. For example, the NSL EA (67) neglected depletion entirely. Thus, contaminants present at the NSL site were assumed to remain at their initial values throughout the duration of all exposure scenarios (some lasting as long as 70 years) considered. This same analysis also posited instantaneous residential development along the periphery of the NSL site—neglecting planning, construction, and occupancy lags. No reason was given for the omission of these factors, although uncertainty about environmental half-lives may have prompted such worst-case assumptions.

Depending on the actual half-lives of the pollutants, the order of the kinetic processes involved, and the occupancy lags, the resulting overstatement in risks could be substantial. For example, assuming first-order kinetics (with decay constant k), an occupancy lag of t_1 years, and a fixed exposure endpoint at t_2 years, the average contaminant concentration C_a, as a fraction of the initial concentration C_1, can be shown by integration of the rate equation to be

$$\frac{C_a}{C_1} = \frac{\exp(-kt_1) - \exp(-kt_2)}{k(t_2 - t_1)}. \quad (1)$$

For first-order kinetics, the rate constant k is related to the half-life, $t_{0.5}$, by means of the well-known relation

$$k = \frac{0.6931}{t_{0.5}}. \quad (2)$$

Calculated exposures and risks (using customary linear models) are directly related to

TABLE 7. Average Pollutant Concentration as Fraction of Initial Value Where First-Order Removal Occurs—Including Effects of Occupancy Lag[a]

Occupancy Lag (years)	Half-Life of Pollutant (years)												
	0.5	1	1.5	2	3	5	7.5	10	15	20			
	First-Order Kinetic Constant (1/years)												
	1.39E+00	6.93E−01	4.62E−01	3.47E−01	2.31E−01	1.39E−01	9.24E−02	6.93E−02	4.62E−02	3.47E−02			
1	2.61E−03	1.05E−02	1.98E−02	2.96E−02	4.98E−02	9.10E−02	1.43E−01	1.93E−01	2.87E−01	3.67E−01			
2	6.63E−04	5.30E−03	1.26E−02	2.12E−02	4.01E−02	8.04E−02	1.32E−01	1.83E−01	2.78E−01	3.58E−01			
3	1.68E−04	2.69E−03	8.08E−03	1.52E−02	3.23E−02	7.10E−02	1.22E−01	1.73E−01	2.68E−01	3.50E−01			
4	4.27E−05	1.37E−03	5.16E−03	1.09E−02	2.60E−02	6.28E−02	1.13E−01	1.64E−01	2.60E−01	3.42E−01			
5	1.08E−05	6.94E−04	3.30E−03	7.85E−03	2.10E−02	5.55E−02	1.05E−01	1.55E−01	2.51E−01	3.34E−01			
6	2.75E−06	3.52E−04	2.11E−03	5.64E−03	1.69E−02	4.91E−02	9.69E−02	1.47E−01	2.43E−01	3.26E−01			
7	6.99E−07	1.79E−04	1.35E−03	4.05E−03	1.36E−02	4.34E−02	8.97E−02	1.39E−01	2.35E−01	3.19E−01			
8	1.78E−07	9.09E−05	8.66E−04	2.91E−03	1.10E−02	3.84E−02	8.31E−02	1.32E−01	2.27E−01	3.12E−01			
9	4.52E−08	4.62E−05	5.55E−04	2.09E−03	8.87E−03	3.40E−02	7.69E−02	1.25E−01	2.20E−01	3.04E−01			
10	1.15E−08	2.35E−05	3.55E−04	1.50E−03	7.16E−03	3.01E−02	7.13E−02	1.18E−01	2.13E−01	2.98E−01			
12	7.42E−10	6.08E−06	1.46E−04	7.78E−04	4.67E−03	2.36E−02	6.13E−02	1.06E−01	2.00E−01	2.84E−01			
14	4.81E−11	1.57E−06	5.99E−05	4.03E−04	3.04E−03	1.85E−02	5.27E−02	9.56E−02	1.87E−01	2.72E−01			
16	3.12E−12	4.08E−07	2.47E−05	2.09E−04	1.99E−03	1.45E−02	4.54E−02	8.61E−02	1.76E−01	2.60E−01			
18	2.02E−13	1.06E−07	1.02E−05	1.08E−04	1.30E−03	1.14E−02	3.91E−02	7.75E−02	1.65E−01	2.48E−01			
20	1.31E−14	2.75E−08	4.20E−06	5.64E−05	8.52E−04	9.01E−03	3.38E−02	6.99E−02	1.55E−01	2.38E−01			
25	1.43E−17	9.57E−10	4.63E−07	1.11E−05	2.98E−04	5.00E−03	2.35E−02	5.42E−02	1.33E−01	2.13E−01			
30	1.57E−20	3.36E−11	5.16E−08	2.20E−06	1.06E−04	2.81E−03	1.65E−02	4.23E−02	1.14E−01	1.91E−01			
40	2.00E−26	4.38E−14	6.78E−10	9.18E−08	1.40E−05	9.25E−04	8.39E−03	2.63E−02	8.52E−02	1.55E−01			

[a] Exposure endpoint = 70 years.

concentrations, so the ratio of the actual risk to that calculated assuming a constant pollutant concentration is equal to C_a/C_i. Table 7 shows this quantity as a function of the half-life and the occupancy lag. For example, this ratio is 2.1×10^{-2} if the exposure endpoint is 70 years, the half-life is 3 years, and the occupancy lag is 5 years, implying that the conservative model overstates risk—considering this factor alone—by a factor of approximately 48. Fortunately, these omissions are readily corrected.

2 Collect Relevant Data on Uncertain Parameters

It is sometimes taken for granted that uncertainty cannot be reduced in risk analysis because key data elements are unknown, and perhaps even unknowable—at least within the time and budget constraints generally imposed on the analyst.

Although this may be true in certain instances, it certainly does not apply across the board. Why, for example, should assumed soil ingestion rates (often a key input to risk analyses) vary as much as the estimates given in Table 4? If there is *no* basis for selection of a most likely value, additional experiments and measurements can be made—and at arguably modest cost compared to potentially inflated cleanup costs of hazardous waste sites if the current worst-case estimates continue to be used.

Other parameters of exposure or risk models that are considered to lack certainty but could easily be measured include the following:

1. Data regarding dermal contact with soils. Realistic data are lacking on human activity patterns involving soil contact. For example, who gardens? How often? What soil contact rates are appropriate? An exhibit analogous to that shown in Table 4 can be prepared for each of these important factors. Unlike some of the more esoteric aspects of potency determination, answers to these questions could be determined by relatively straightforward surveys.
2. Data regarding the absorption (bioavailability) of chemicals through ingestion, dermal contact, and respiration. Uncertainty exists for these parameters (e.g., reported dermal absorption fractions for PCBs from soils or dusts range from 7×10^{-4} to 5×10^{-1} in the literature).

A salutary development in this regard is the EPA's recent decision to sponsor additional research on soil ingestion and to suggest additional research on bioavailability (68). The value of acquiring additional information about uncertain parameters can sometimes be treated analytically using the methods of decision theory (69). These techniques can be used to identify cost-effective approaches to uncertainty reduction.

3 Risk Assessors Should Understand the Spirit of Legislative Action

A third idea is that the (interpretation of the) legislative mandates of regulatory agencies should be examined to identify potentially counterproductive aspects. With respect to PCBs, for example, the FDA established tolerance levels for PCBs in fish in the context of its statutory framework. As the FDA notes (70):

> Section 406 of the Federal Food, Drug, and Cosmetic Act ("the act"), 21 U.S.C. 346, authorizes the establishment of tolerances for poisonous or deleterious substances added to food that cannot be avoided by good manufacturing practice. PCBs are such a substance. Although *the agency's paramount concern is protection of the public health*, under section 406 *the agency must consider, in establishing a tolerance, the extent to which a contaminant is unavoidable*. In essence, the agency is permitted to find where the proper balance lies between adequately protecting the public health and avoiding excessive losses of food to American consumers. 44 FR 38330-31. [emphasis added]

Put somewhat differently, tolerance levels are established at a level "appropriate to protect the public health" or to "provide an adequate degree of public health protection." But tolerances established by the FDA also reflect existing levels of contamination and the extent of its "avoidability" in food products to be regulated.

On first reading, the "balancing provisions" of the Food, Drug, and Cosmetic Act (Section 406 of 21 USC 346) appear quite reasonable. But on more careful examination, there are curious, and arguably perverse, consequences of the FDA's present interpretation of this legislative mandate.

Consider, for example, two hypothetical foodstuffs, A and B, each contaminated initially to an identical degree with the same hazardous substance:

1. In product A, the contamination levels are expected to remain constant over time.
2. In product B the levels of contamination are expected to decline in the future.

Assuming that products A and B are consumed in equal amounts in the human diet and are absorbed equally, the lifetime incremental health risks associated with consumption of product B are obviously smaller. Product B, by any objective standard, presents less of a health hazard than product A. Yet, there is no guarantee that FDA tolerance levels for the hazardous contaminant in product B will be larger than, or even the same as, those for product A. In fact, quite the reverse is likely to be true. This is because the risks associated with product B become progressively more "avoidable" over time—a phenomenon that allegedly justifies lower tolerance levels.

The above situation is by no means hypothetical; it has occurred with respect to PCBs in poultry and fish. In 1977, the FDA (71) proposed a reduction in the tolerance level for PCBs in poultry (later implemented) from 5 ppm (fat basis) to 3 ppm (fat basis), not because PCBs were thought to be more dangerous, but rather because elevated PCB levels were *infrequent* and *declining* in poultry:

> Because the frequency of PCB residue occurrence in feeds is low, the likelihood of residues in poultry reaching the 3 ppm (fat basis) level is very small. Moreover, data regarding PCB residues in poultry confirm this and show that PCB contamination of poultry is very sporadic and infrequent. As such, this food is not a significant source of dietary PCBs. A tolerance of 3 ppm (fat basis) will continue to provide this assurance, while also providing adequate protection for the consumer. Therefore, the Commissioner proposes to reduce the temporary tolerance for poultry from 5 ppm to 3 ppm (fat basis). As stated previously, the finished feed tolerance of 0.2 ppm cannot be reduced at this time because the analytical methodology necessary to enforce a lower tolerance is not available. The Commissioner advises that when such methodology becomes available so that the 0.2 ppm feed tolerance can be reduced, the tolerance for PCB residues in poultry will also be reevaluated.

Likewise, with respect to fish, the FDA concluded that declining PCB levels were a reason for *reducing* tolerances (72): "Based on the declining incidence of PCB contamination, which means that PCBs are now avoidable in food to a greater degree now than they were earlier... FDA decided the PCB tolerances should be reduced." Later in this same document, in response to the comment that PCB levels in fish were declining, the FDA reaffirmed its proposed standard, noting (73): "Moreover, that PCB levels are declining (i.e., that PCBs are becoming more avoidable) is a reason to consider lowering the

tolerance, not a justification for leaving it unchanged." Certainly, it is time to rethink this interpretation.

4 Incorporate Consistency and Plausibility Audits into Modeling Efforts

A fourth idea is to subject candidate risk and exposure models to more searching inquiry—to conduct a consistency or plausibility audit on the model.

Although experimental measurements may be considered the ultimate validation of assumptions, these are not always possible and other approaches may be necessary. In some cases, simple material balances can furnish useful consistency and plausibility checks on the adequacy of an exposure–risk model. For example, as noted, a risk analysis was conducted to help determine appropriate reentry guidelines for PCB- and TCDD-contaminated surfaces at the Binghamton (NY) State Office Building (74). The risk analysis for contaminated surfaces assumed, among other things, that:

1. Workers (50 kg) would labor in the building for 250 days/yr over 30 years.
2. The worker's total body surface area is 1.46 m^2, with the hands accounting for 4.5% (0.066 m^2) and the arms accounting for 19% (0.28 m^2) of this total.
3. The worker is assumed to ingest the contamination from an area the size of 5% of his/her hand (0.0033 m^2) every workday.
4. The worker is assumed to make dermal (bare skin) contact with an area 25% that of his/her arms (0.0694 m^2) every workday.
5. (Unstated) All PCBs are transferred from walls upon contact with contaminated surfaces.

Given these assumptions, the daily PCB intake was calculated by Kim and Hawley (74) by the following equation (with slight differences in notation):

$$\text{intake } (\mu g/\text{day}) = C_{sr} f_{GI}(0.0033) + f_{dl}(0.0694) \tag{3}$$

where C_{sr} = residual surface contamination level ($\mu g/m^2$)
f_{GI} = gastrointestinal absorption fraction
f_{dl} = dermal absorption fraction.

The risk analysis proceeds from the exposure calculation given by equation (3).

Upon initial review, the approach appears plausible. However, a "reality check" would have revealed that it was unrealistic. A simple example illustrates the problem. Assume that the hypothetical building occupant works in a one-person office of dimensions 8 ft × 10 ft × 12 ft. The total area of the walls of this office is 352 ft^2 (327,008 cm^2). Assuming, for illustrative purposes, a 100 $\mu g/100\,cm^2$ level of PCB surface contamination, the total amount of PCBs on the walls is

$$\frac{100\,\mu g}{100\,cm^2} \times 327{,}008\,cm^2 = 327{,}008\,\mu g.$$

Now according to the Kim–Hawley model, PCBs are contacted and removed by two human features, arms and hands. Using the above factors, the total amount of PCBs

removed each day by incidental skin contact with the arms, denoted X, would be

$$(1.46\,m^2 \text{ body surface}) \times \left(\frac{0.19\,m^2 \text{ arm surface}}{m^2 \text{ body surface}}\right) \times \left(\frac{0.25\,m^2 \text{ contacted}}{m^2 \text{ arm}}\right)$$
$$\times (1.0 \text{ transfer fraction}) \times (10{,}000\,cm^2/m^2) \times \left(\frac{100\,\mu g \text{ PCB}}{100\,cm^2}\right),$$

or

$$X = 693.5\,\mu g.$$

Similarly, the amount of PCBs removed each day by skin contact with the hands, denoted Y, is

$$(1.46\,m^2 \text{ body surface}) \times \left(\frac{0.045\,m^2 \text{ hand}}{m^2 \text{ body}}\right) \times (1.0 \text{ fraction transfer})$$
$$\times \left(\frac{100\,\mu g \text{ PCB}}{100\,cm^2}\right) = Y = 657\,\mu g.$$

The total removed per day = $X + Y = 1350.5\,\mu g$. It should be noted that not all of this amount of PCBs would be absorbed, but all would be removed. (The above calculation assumes that the entire surface area of the contaminated surface will be contacted. Any corrections for inaccessible areas would reduce the estimated amount of PCB uptake and removal.)

Assuming that these rates continue (as is done in the Kim–Hawley analysis), the total number of days required to exhaust the PCB contamination at the surface is (32,7,008 μg) × (1350.5 μg/day) = 242 days. But the exposure scenario used to estimate the cancer risk assumes a 30-year period, 5 days/wk, 50 wk/yr. Clearly, the assumptions in the exposure estimate were unrealistic, and had a plausibility check been done, this could have been identified early in the analysis process.

It is interesting that the authors considered various adjustments (first-order decay) to model volatilization or other (unstated) depletion mechanisms, but no consideration was given to the process of depletion inherent in the mechanism of exposure itself. For these and other reasons, the Kim–Hawley analysis must be regarded as unrealistically conservative.

5 Use Computational Approaches That Avoid "Catch All" Assumptions

A fifth idea is to design models insofar as possible to avoid "catch all" parameters that may be ambiguous. Alternative risk and exposure models can differ in the extent to which subjective elements can enter the analysis process. Figure 1, for example, diagrams the logic of an EPA risk analysis designed to estimate appropriate cleanup levels for indoor PCB spills (75). This figure also contains the EPA's numerical estimates purporting to show that residual surface PCB contamination levels of 100 $\mu g/100\,cm^2$ result in lifetime incremental health risks of the order of 10^{-4}. The logic behind the model is depicted in Fig. 1.

This analysis assumed that a notional room (of area 438,000 cm^2) has been decontaminated by replacing so-called high contact areas (area 27,871 cm^2) and cleaning

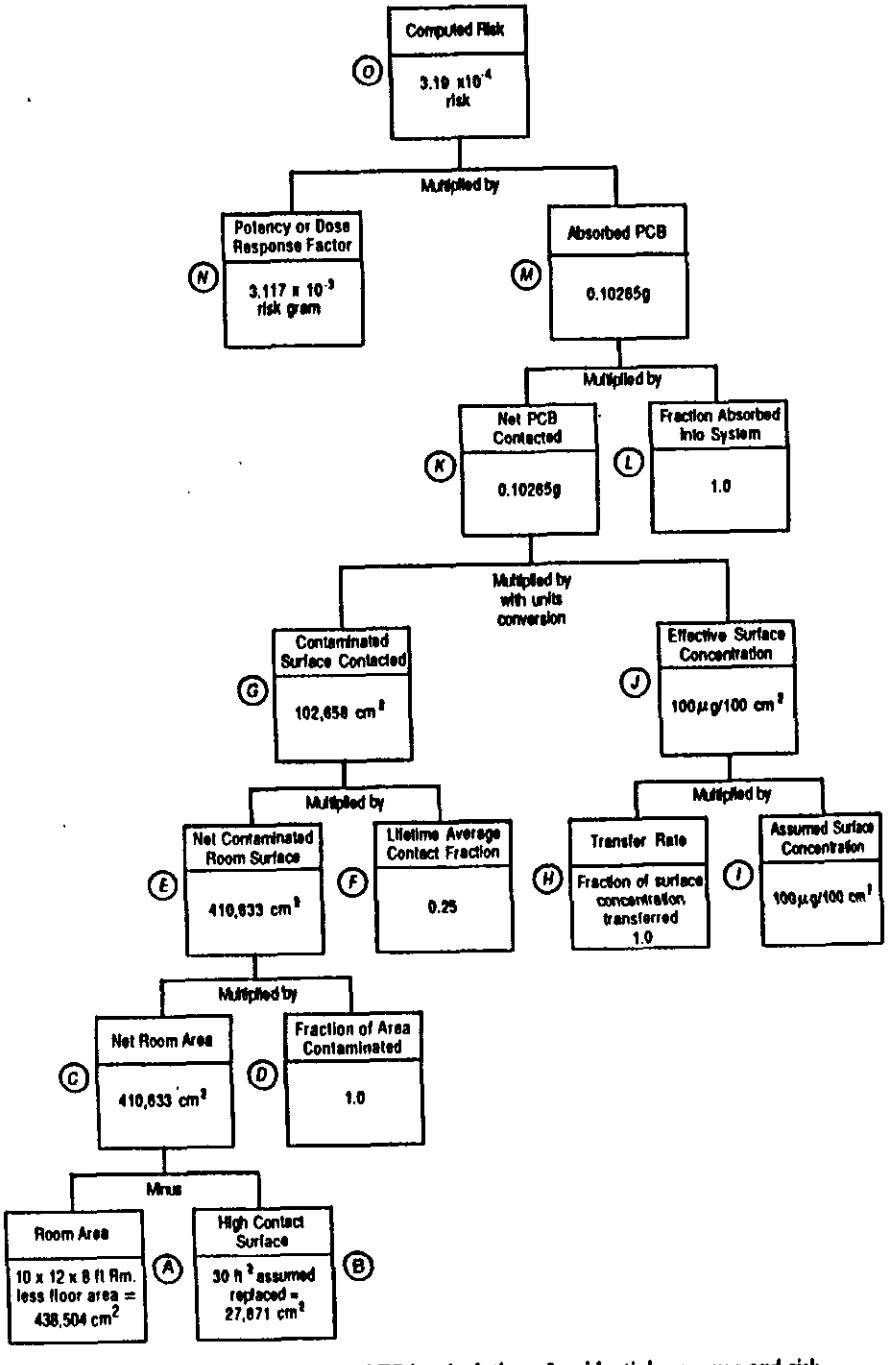

Figure 1. Schematic diagram of EPA calculation of residential exposure and risk.

the remaining surface areas to a residual PCB concentration of 100 μg/100 cm². After cleanup, the net room area (box C in Fig. 1) is calculated as the difference between the original contaminated area (box A) and the high contact area (box B). All of this net area is assumed to be contaminated (box D), so the net contaminated room surface (box E) is the same as the original area minus the replaced high contact areas.

Human occupants are assumed to have incidental skin contact with these contaminated surfaces. Based on the assumption of an assumed residual surface PCB concentration of 100 μg/100 cm² (box I) and the assumption that, upon contact, 100% of the surface concentration is transferred to exposed skin (box H), the potential amount of PCBs transferred (effective surface concentration, box J) per 100 cm² contaminated surface contacted can be calculated. No surface depletion mechanisms (e.g., volatilization, penetration into wells) or effects of encapsulation (e.g., by painting) were considered in this EPA analysis.

A key computational artifice employed in the EPA model is the lifetime average contact fraction (LACF), defined as the fraction of the contaminated area that would be touched by the room's occupant in a lifetime of exposure, which was set equal to 0.25 in the analysis (box F). Multiplication of the LACF by the net contaminated room surface (box E) enables calculation of the contaminated surface contacted (box G). In turn, this quantity multiplied by the "pickup rate" or effective surface concentration (box J) estimates the net PCB contacted in a lifetime (box K), numerically equal to approximately 0.1 g. All this PCB is assumed to be absorbed (box L) even though studies (76) point to absorption fractions no more than half this amount for PCB liquids, and appreciably less for PCBs in soils or household dust (77). Finally, the potency of the PCBs is factored in (box N) using a potency value smaller than the largest given in Table 2.

The assumptions in this analysis are obviously quite conservative, but the point of introducing this example has to do with the computational construct rather than the numerical inputs. The LACF construct in the model displayed in Fig. 1 obviously simplifies the model materially. Use of this model, for example, does not require the explicit specification of human activity patterns (i.e., determination of contact frequencies) or of surface depletion mechanisms (e.g., volatilization, adsorption into interior surfaces, photolysis if exposed to sunlight). But it also leaves the analyst in a quandry when it comes to estimation of the LACF. Spend a few minutes trying to estimate your LACF for the room in which you are seated as you read this. Surely, at some time in your life, you *could* come into contact with every portion of the wall or ceiling, even those that are nominally "masked"—knocking about to retrieve your favorite pipe that fell behind the couch, replacing the ceiling light fixture, spring cleaning, mopping up a spill under the refrigerator, rehanging pictures, or painting. Nonetheless, as a practical matter, only a small portion of the room's surface area is likely to be touched with any frequency. Although the LACF concept simplifies the problem numerically, it only masks the real complexity of the physical situation being studied. The model permits the risk analyst to "doodle" and conduct a fortiori (but even if) analysis, but in the end this formulation lacks utility. Moreover, such an ambiguous construct invites a subjective rather than reasoned response. It is somewhat surprising that EPA chose a value as "low" as 0.25.

This example illustrates the linkage between the complexity of the calculated approach and that of preparing inputs. The simplistic construct depicted in Fig. 1 removes computational complexity at the expense of realism and ease of determining inputs. A balance must be struck among competing objectives, such as simplicity, realism, and availability of data, if a useful and credible risk estimate is to be produced.

6 Provide Clear Guidance to Interested Parties

If the risk anslysis community needs to emphasize realism in analysis, the users of analysis (policymakers and public) also need to understand the issue to interpret properly the results of analysis. This places a burden on the analyst to communicate clearly. As the example below shows, this is difficult.

As noted, the FDA undertook a risk analysis in support of its decision to revise the tolerance level for PCB-contaminated fish downward from 5 ppm to 2 ppm. A critique of this analysis highlighted points of conservatism and concluded that the FDA analysis was likely to have overstated risks by orders of magnitude (78). Although the FDA did not quantify the possible magnitude of overstatement of risks, it was open in acknowledging the uncertainties and the judgmentally conservative character of its resolution. For example, in the background to its 1979 ruling (79), the FDA stated the following:

> Hence, in deciding the appropriate levels for PCB tolerances under section 406, FDA *had to make some extraordinarily difficult judgments.* It has had to decide, in effect, where the proper balance lies between providing an adequate degree of public health protection and avoiding excessive losses of food to American consumers. [emphasis added]

The FDA also noted that (80) "it [FDA] also must make that judgment on the basis of *data that are incomplete, or even in dispute, and that can easily lead reasonable people to different conclusions*" [emphasis added].

The FDA acknowledged uncertainties regarding the carcinogenicity of PCBs in humans (81):

> FDA considers the question of the carcinogenicity of the PCBs unresolved. For the purposes of this risk assessment on PCBs, however, the agency treated the various PCBs as though they were carcinogenic and it considers the carcinogenicity of PCBs to be a matter worthy of further serious inquiry.

Having thus dealt with this key question by assumption, the FDA risk analysis proceeded to incorporate other conservative assumptions. These, too, were explicitly acknowledged by the FDA (82):

> The risk assessment the agency made incorporated several conservative assumptions that were designed to avoid understatement of the human risk. *Thus, it is expected that the actual risk experienced by consumers of the 12 more heavily contaminated species covered by the risk assessment is less than that estimated.* Moreover, the average consumer, who eats fish from a variety of freshwater and marine sources, will actually experience a far lower level of PCB exposure and a correspondingly lower degree of risk than those whose fish consumption is concentrated among the more heavily contaminated (predominantly freshwater) species. [emphasis added]

This statement was echoed in the 1979 *Federal Register* notice (83):

> *These risk assessment methods do not purport to quantify precisely the expected human risk, but rather attempt to estimate in quantitative terms an upper limit on the risk to humans that can be expected from a given level of exposure* to a toxic substance, assuming humans are no more susceptible to the effects of the substance than are the most susceptible members of the animal species for which toxicity data are available. These risk assessments can be useful as a means of comparing risks at various exposure levels and illustrating the toxicological judgment that a reduction in exposure will reduce risk. *Because of all the problems inherent in extrapolating from animal data to the expected human experience, however the numbers produced by a risk assessment must be interpreted cautiously: They are estimates of upper limits on risk* and, though potentially useful for comparative purposes, *cannot be said to quantify actual human risk* precisely. These assessments attempt to avoid underestimating human risk.... [emphasis added]

and again in this same FDA document (84):

> As explained in the report (Ref. 45), *the utility of this risk assessment for evaluating actual risk to humans from exposure to PCBs is extremely limited.* This is due both to difficulties inherent in making such extrapolations from animals to humans and, perhaps more importantly in this instance, to gaps and uncertainties in the data available for this particular risk assessment. For example, the toxicity studies on which the risk assessment is based used commercial preparations of PCBs, which are chemically different from the PCB residues found in fish and which contain small amounts of highly toxic impurities (e.g., dibenzofurans) not known to be present in fish residues. Also, in making the exposure estimates required for the risk assessment, it was necessary to use existing data on the numerical distribution of PCB levels in fish and rely on the assumption that the effect of a given tolerance level is to remove from commerce all fish containing PCBs exceeding the tolerances. It is possible that neither the assumption nor the data precisely reflect what actually occurs.
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> *For these reasons and others discussed in the report (Ref. 45), the risk assessment does not provide a basis for precise quantification of the amount of risk reduction accomplished by reducing the fish tolerance.* [emphasis added]

Notwithstanding such a measured appraisal of the uncertainties that clouded the standard-setting process, this standard, once established, was viewed as absolute. After a 5-ppm (now 2-ppm) standard was established, regulators, particularly in states that adopted the FDA action limit as a state standard, often acted as though consumption of fish contaminated with 4.8 ppm was "safe" but consumption of even one fish with 5.1 ppm was "unsafe." Thus, disclaimers, caveats, and qualifiers may not be sufficient to help users understand the limitations of a risk analysis. Nonetheless, it is clear that the risk analysis community needs to communicate more effectively.

Two approaches may have merit in this connection:

1. The first is what might be termed the "comparative risk" approach popularized by Crouch and Wilson (85), Ames et al. (86), and others. This approach presents estimated risks in the context of other related and everyday risks. The objective is to provide a frame of reference to help interpret the risk estimates. In my experience the comparative risk analysis is useful, but limited. The work of Slovic (87) demonstrates that public perceptions of risk are often different from that of "experts."
2. The second idea is to portray uncertainty in quantitative terms, as is illustrated below.

7 Undertake Sensitivity Analyses

It is suggested above that attention be directed toward the development of most likely or expected risks. "Conservative" estimates can be placed in perspective by making replicate computations with alternative sets of assumptions. This serves to identify the sensitivity of the calculated result to the various assumptions—and thus identify key uncertainties—as well as to bracket potential risks so that the policymaker can make a more informed choice. Other formal tools of statistics can also be applied.

Regulatory agencies appear to be increasingly receptive to sensitivity analysis or the use of multiple assumption sets. For example, in 1980, in response to comments

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15

Risk Analyses of Buried Wastes from Electricity Generation*

Bernard L. Cohen

Department of Physics, University of Pittsburgh, Pittsburgh, Pennsylvania

1 INTRODUCTION

The straightforward way to do a probabilistic risk analysis (PRA) is to identify all possible sequences of events that can lead to deaths among the public (or to some other targeted endpoint), estimate the probability for each event, multiply these probabilities for all events in a sequence to determine the total probability for that sequence, and finally add up the probabilities for all sequences. Interdependencies of events (common mode failures) must be taken into account, which can add greatly to the complication and uncertainty (1). Such a PRA involves development of a large program run on a digital computer.

This type of PRA has been carried out for systems like aircraft and nuclear power plants where there is complete knowledge of construction details and failure-rate experience with every component. But even in these cases, many approximations must be made, and uncertainties are quite large. If one were to apply this technique to wastes buried in the ground, the difficulties would be enormously greater because knowledge of the system is much less complete and is changing with time. Geochemistry is a much more complex subject than mechanical or electrical design of machines, and it is sensitive to a number of factors on which there is limited information.

An alternative approach is to replace the digital computer with an analog computer. Constructing such an analog computer would be a tremendous project, and it would be enormously expensive. However, that analog computer is now available, and we are all free to use it. It is our earth itself.

This chapter describes how such an approach can be used. For example, to study how waste converted into rock behaves, how ordinary rock behaves will be reviewed. Several cases of this type directed at the analysis of wastes generated by nuclear and by coal-burning power plants are presented.

*Portions of this chapter have been published previously in *American Journal of Physics*, Vol. 54, p. 38 (1986).

The New York Times

Late Edition
New York: Today, mostly sunny, cool. North winds, 10-20 m.p.h. High 74. Tonight, clear, cooler. Low 57. Tomorrow, sunny. High 77. Yesterday, high 89, low 77. Details are on page 39.

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gton IRT: to Disaster

By ...EN

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...irting With Danger

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...all its heavy responsibility, ...was an anonymous one: ...y, one of 2,900 New York ...train operators — com... ...referred to as motormen, ...ough more than 100 are ... — was glimpsed fleeting... ...t all, in the tiny cabs of ...entering stations, but was ...ed daily with the smooth ...on of multi-million dollar ...and the safety of tens of ...assengers. ...tential for catastro... always present, experts

...ued on Page 30, Column 1

EXPERTS QUESTION STAGGERING COSTS OF TOXIC CLEANUPS

A NEW VIEW OF THE PERILS

Most Health Dangers Could Be Eliminated for a Fraction of Billions Now Estimated

By PETER PASSELL

A decade after Washington declared war on businesses that expose the public to hazardous wastes, environmental experts are questioning the unquestionable: Is it worth spending a staggering $300 billion to $700 billion to restore waste sites to pristine condition?

While acknowledging that no level of exposure to dangerous chemicals is desirable, the experts argue that the risks should be put in perspective.

Virtually all of the risk to human health, most analysts agree, could be eliminated for a tiny fraction of these sums. In a typical project, in Holden, Mo., $71,000 would be enough to isolate an abandoned factory containing residues of toxic chemicals, making it extremely unlikely that anyone would ever be harmed by the wastes. Another $3.6 million would clean up virtually all residues and bury remaining traces under a blanket of clay.

What Is Justified?

But state and Federal laws requires cleanup that would cost $13.6 million to $41.5 million. "The last couple turns of the screw cannot be justified on economic criteria," said Tom Grumbly, an environmentalist who is president of Clean Sites, a nonprofit organization in Virginia that advises communities on hazardous waste cleanups.

To be sure, the problem of weighing cleanup benefits against the costs is complicated by a lack of information about how dangerous individual chemicals are, and in what concentrations. Nonetheless, experts insist that what began as a crusade against polluters has become a diversion, siphoning money and technical expertise from more pressing environmental concerns.

Tougher Criteria Suggested

More stringent scientific criteria should be used to identify waste sites needing immediate attention, they say. And once identified, the cleanup should be carefully aimed at saving lives rather than restoring land to preindustrial condition.

Analysts acknowledge that redirecting Government policy will be nearly impossible without a radical change in

Continued on Page 28, Column 1

Soviets Prepare to Design a New System

Associated Press

Revelers celebrating in Moscow yesterday as legislators prepared to meet this week to begin debate on a new system of government.

Debate Expected on Transition and Baltics

By BILL KELLER

Special to The New York Times

MOSCOW, Aug. 31 — As two more republics joined the mass exodus from the dissolving Soviet Union, the nation's legislature set the scene today for a crucial Congress of People's Deputies to debate a new system of government after the collapse of Communist authority.

The congress, the highest constitutional authority in the faltering union, is to open Monday with an agenda that includes a report by President Mikhail S. Gorbachev on his vision of a new Soviet community, the composition of a recast legislature to preside over the transition, and an appeal from the Baltic republics to formally recognize their freedom.

While the members of the Supreme Soviet, the country's standing legislature, dithered to adjournment in advance of the opening of the much larger and more powerful congress, the mood of political anxiety generated by two weeks of national upheaval slackened into festivity.

More Casual Air Prevails

Muscovites strolled to a riverside festival of pop music and comedy skits, an annual event that this year became a celebration of victory over Communism.

This afternoon, Mr. Gorbachev startled Saturday strollers by emerging from Moscow City Hall with Mayor Gavriil K. Popov and a flotilla of bodyguards, and walking a mile to the Kremlin. He did not chat with passers-by, who nonetheless reacted with applause and shouts of support.

There were other light moments. Newspaper readers chuckled over accounts of the prison routine of the failed junta that, less than two weeks ago, seemed to hold the country in a state of neo-Stalinist paralysis.

Staff Will Operate Pravda

Pravda, long the voice of Communist Party orthodoxy, reappeared after a weeklong suspension for its service as a mouthpiece of the failed putsch. Now under the management of its staff, the humbled newspaper promised to become the voice of "civic accord."

"We will strive to work in such a way that the next junta, if it ever comes, will close Pravda first," a staff commentator wrote.

Today, the Central Asian republics of Uzbekistan and Kirghizia joined the parade of constituent republics declaring their independence. They were the seventh and eighth republics to end their membership in the union since the coup. Two others, Lithuania and Georgia, asserted their independence earlier, and Armenia has scheduled a referendum on secession next month.

For the Central Asian republics, the declarations did not reflect a desire to go it alone, but a need to position themselves as equals before negotiating the terms of a new relationship.

Uzbekistan, a cotton growing region ...

Aides Say Bush Is Ready to Grant Recognition to Baltics Tomorrow

By ANDREW ROSENTHAL

Special to The New York Times

KENNEBUNKPORT, Me., Aug. 31 — President Bush will extend formal recognition to Latvia, Lithuania and Estonia on Monday regardless of whether the Soviet legislature acts to grant Baltic independence by then, Administration officials said today.

The officials said that although Mr. Bush had been withholding formal recognition of the three republics, out of deference to President Mikhail S. Gorbachev, he had decided that he is giving Soviet legislators enough time to act.

"We wanted to give them a respectable grace period so if we had to act on our own, nobody could complain that we did it precipitously," one official said.

The American officials' comments today appeared to be a clear effort to prod Soviet legislators. The Soviet Congress of People's Deputies meets in Moscow on Monday. It would have a full working day to act even if Mr. Bush were to make such an announcement as early as mid-morning on Monday here, because Moscow time is seven hours ahead of Eastern Daylight Time.

In Moscow, however, there were indications that the huge assembly, which is facing a long agenda of tough issues, would be unable to take up the Baltic issue that soon.

The expected announcement from Mr. Bush will have some flavor of an anticlimax, since the President has acknowledged that it was inevitable and because much of West has already moved to give the Baltic republics formal recognition and establish diplomatic relations with them.

...rl Harbor, Still Elusive

By ...LD

... the Japan-America Society of ... "The time has come for rec...on."

Mr. Inouye, a Democrat who ... right arm as an American ...ghting in Italy, should have to ...ch a plea served only to empha... ...tense state of Japanese-United ...elations today.

...er government appears willing ...e Pearl Harbor ceremonies to ... with the issues that divide ... July the Bush Administration, ...ressure from veterans groups,

...nued on Page 22, Column 1

Women's Tennis, She's Over the Hill at 21

By HARVEY ARATON

...four years of high school in ...Valley, Calif. and three years

Becker Upset at the U.S. Open

the way Americans think about the risks from hazardous waste. Some would try to temper the zeal for large-scale cleanups by asking the communities that benefit to share in the costs. "Everybody wants a Cadillac as long as someone else is paying," said Robert Hahn, an economist at the American Enterprise Institute, a research organization in Washington.

Little Confidence in Washington

But there is little confidence in Washington that cost-sharing would make a big difference in public attitudes. And no enthusiasm can be detected for asking voters to pay to protect themselves against hazards they did not create.

A first priority, argues Frank Blake, a former general counsel to the Environmental Protection Agency who now works for the General Electric Company, is more credible information that lifts the "fog of fear" from public perceptions. Specifically, he says, people need ways to compare the risks of exposure with the dozens of other risks that are accepted as part of daily life.

$11 Billion Is Spent

The best-known Federal waste initiative is Superfund, which has spent $11 billion in a decade on emergency measures at 400 abandoned sites and full-scale cleanups at 60 others. But the cleanup mandated by the Superfund legislation has barely begun.

Some 1,200 other sites are already on the Environmental Protection Agency's national priority list, and the Congressional Office of Technology Assessment expects thousands to be added. What is more, a slew of Federal laws require the eventual cleanup of tens of thousands of other sites polluted by government and business. Mr. Grumbly of Clean Sites says the bill for waste containment could reach $20 billion a year by the end of the decade.

What the Country Can Afford

The country could afford such sums, everyone agrees, if the problem demanded it. Businesses and taxpayers have managed to cope with other anti-pollution regulations that now cost $115 billion annually, by the E.P.A.'s reckoning. But little hard evidence exists to support a crash effort.

People living or working near identified toxic waste sites naturally worry that the chemicals will find their way into air, food and water. But many analysts believe the immediate dangers have been exaggerated.

"When you look for deaths from hazardous wastes, you just don't find them," said Bill Ralston, an analyst at SRI International, a consulting company in California.

The Environmental Protection Agency estimates that roughly 1,000 cancer cases annually can be linked to public exposure to hazardous waste. That hardly makes abandoned waste sites a non-problem in anyone's book.

Many analysts say pollution perils have been exaggerated.

But the environmental agency does rank hazardous waste far behind cancer risks like exposure to chemicals in the workplace and depletion of the atmospheric ozone layer. Moreover, many of these greater risks could be reduced at a tiny fraction of the cost of the hazardous waste fix mandated by law.

The Example of Radon Gas

The E.P.A. estimates, for example, that 5,000 to 20,000 lung cancer deaths are caused each year by indoor exposure to radon gas leaking from underground rock formations. Most of these deaths could be prevented by performing inexpensive tests on buildings in radon-prone areas and ventilating basements where concentrations of the radioactive gas are high.

What explains this apparent distortion in environmental priorities? Political economists have long noted that the focused convictions of the few almost always dominate the weakly held views of the many. And Christopher Daggett, the former head of the New Jersey Department of Environmental Protection, argues that nothing focuses convictions like a nearby waste site.

for The New York Times

A view of PCB contaminated buildings on the property of the Rose Chemical Company in Holden, Mo. Experts on environmental issues insist that what began as a crusade against polluters has become a diversion, siphoning money and technical expertise from more pressing environmental concerns.

The Big Cleanup

The cost of cleaning up several categories of polluted sites.

Category	Number of sites (estimated)	Estimated cost ($ billions)
Superfund abandoned sites	4,000	$80-120
Federally owned sites	5,000-10,000	75-250
Corrective action on active private sites	2,000-5,000	12-100
Leaking underground storage (tanks)	350,000-400,000	32
State law mandated cleanups	6,000-12,000	3-120+
Inactive uranium tailings	24	1.3
Abandoned mine lands	22,300	55

Source: Robert Hahn, American Enterprise Institute, Office of Technology Assessment

"Try telling people that the leaking drums across the street aren't a hazard," he said.

Legal and Moral Responsibility

There are the questions of legal and moral responsibility. Some environmental hazards, like auto emissions, are everyone's fault — and therefore no one's. Some, like cigarette smoking, are no one's fault but one's own. But Mr. Gough notes that the public blames "greedy and thoughtless" corporations for the waste peril and is thus little inclined to weigh the benefits of cleanups against the costs.

It is not surprising, then, that communities with waste sites in their midst want them restored to how they were before industrial use. Nor is it surprising that the elected officials and administrators who write the rules for cleanups take their cues from an angry and anxious public.

Consider the 11-acre Rose Chemicals Company site in Holden, Mo. Until 1986 the site was used to store and process PCB's, toxic liquid chemicals prized by makers of electrical transmission equipment for their insulating properties and their stability at high temperatures. The barrels of PCB's were removed long ago, but residues remain in the buildings, the soil and the bed of a small stream.

A Range of Options

An E.P.A. analysis says that for $71,000, the site could be permanently isolated from the community. The town's drinking water would be safe because there is no groundwater to tap near the site. But under Superfund regulations, this cheap fix would be unacceptable because there is one chance in 10,000 that someone would develop cancer from eating the cattle that grazed near the property's perimeter — and roughly the same risk that a determined trespasser could get cancer from repeated contact with building surfaces.

Other possible fixes range from cleaning the stream bed and capping the site with 30 inches of clay ($3.7 million) to removing 14,000 tons of contaminated soil and building materials and incinerating it elsewhere ($41.5 million). The E.P.A.'s preferred $13.6 million option: remove all of the suspect soil and materials, then incinerate the most contaminated debris and bury the rest in a specially designed landfill.

A Cheaper Alternative

Mr. Grumbly of Clean Sites notes that the E.P.A.'s compromise would allow virtually any future use of the site. And he points out that it would cost just a third as much as the maximum effort.

But others may wonder whether the more flexible use of the land is worth the extra $800,000 an acre beyond the cost of scrubbing the stream and capping the property. And still others may ask whether a bare-bones fix — one that reduces neighborhood cancer risks to, say, one-thousandth the chance of getting cancer from a lifetime of normal exposure to the sun — would not be adequate.

Mr. Hahn of the American Enterprise Institute and others inclined toward less ambitious goals suggest using financial incentives to discipline the decision-making process. If those at risk from hazardous waste sites wanted more than a basic cleanup, they could be asked to share in the cost.

Dan Dudek, an economist at the Environmental Defense Fund, a mainstream environmental group, offers a variation based on the carrot rather than the stick. Instead of taxing communities to cover the extra cleanup effort, he would give them part of the savings associated with a less-than-total repair. The town of Holden, for example, might be offered 20 percent of the $9.9 million difference as an incentive to save lives in other ways — say, by purchasing more efficient fire-fighting equipment, or by testing every home for radon and lead contamination.

That idea appeals to Francis Brillhart, the mayor of Holden, who said he fears that the "E.P.A. is going a bit overboard," slowing the cleanup by choosing a method that requires incineration. If Holden could keep some of the savings generated by a faster cleanup, he says, so much the better.

But few experts think many communities would be likely to take the financial bait. The approach presumes that people would compromise on safety in return for lower taxes or improved public services. Everyone trades safety for money or convenience or simply pleasure in daily life — new tires are safer than worn ones, turkey breast is safer to eat than a cheeseburger. But people rarely acknowledge they are making choices, even to themselves.

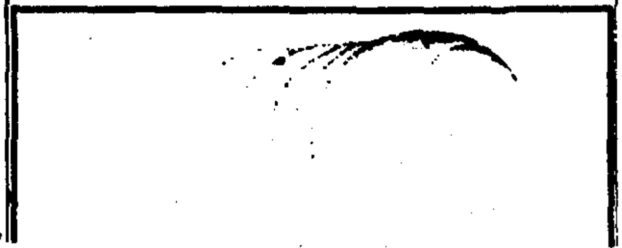
Need to Educate the Public

That is why most analysts worried about the open-ended commitment to scrubbing the country pin their hopes on educating the public about relative risks. "We need more and better information about health risks," said Katherine Probst, an analyst at Resources for the Future, a nonprofit environmental research group.

The way in which the information is presented may also matter: knowing hazardous waste can kill is less useful than knowing it kills one-twentieth as many Americans as radon.

Mr. Grumbly of Clean Sites argues that the credibility of government turns on the transparency of its decision-making. One key to a rational public debate, he insists, is to "let the public in on the game."

But Mr. Grumbly is an optimist. "People understand," he says, "when they are wasting other people's money."



HEALTH RISK ASSESSMENTS: OPPORTUNITIES AND PITFALLS

DENNIS J. PAUSTENBACH

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Volume 14, Number 2

Health Risk Assessments: Opportunities and Pitfalls

Dennis J. Paustenbach*

I. Introduction

To fulfill my role in this symposium, I will discuss the regulated community's thoughts regarding current approaches to health risk assessments conducted by regulatory agencies within the United States. It might be more appropriate to say that I will discuss some of the scientific shortcomings which have crept into the practice of risk assessment and how regulatory agencies and scientists are working to overcome them. These shortcomings, more often than not, force risk assessments to overstate the likely human health risks associated with exposure to low levels of environmental pollutants.[1]

Environmental consulting firms typically serve the regulated community and its lawyers, solving problems involving contaminated soil, contaminated groundwater, airborne emissions, or a need for an operating permit. Generally, the chemicals are carcinogens, developmental or reproductive toxicants, or are highly persistent in the environment. The firms are frequently at odds with a regulatory agency. Also, they are often involved in litigation over the degree of necessary clean-up. More often than not, personal injury claims have been filed which allege that health has been or is likely to be affected due to exposure to contaminated soil, air, or water. The consulting firm's role is to assist corporations and their attorneys by developing a more thorough, balanced and therefore, credible health risk assessment than that put forward by the government or a plaintiff's attorney.

* Vice-President, McLaren Environmental Engineering, Inc., and head of the ChemRisk Division, Alameda, Ca. An earlier version of this article was presented as a paper at the Symposium on Risk Assessment presented by the Environmental Law Committee of the Association of the Bar of the City of New York on February 4, 1988 in New York City.

1. Ames, *Six Common Errors Relating to Environmental Pollution*, 7 Reg. Toxicol. & Pharm. 379, 380 (1987).

II. THE NEED FOR RISK ASSESSMENTS

Risk assessment has been a topic of intense interest during the 1980's.[2] The development of a human or ecological risk assessment can be a complex undertaking which requires the assimilation and interpretation of large quantities of scientific and medical data.[3] Although a number of definitions have been offered in the literature, it is acceptable to say that risk assessments are a way of using existing toxicity, epidemiology, environmental fate, and exposure information to describe the likely health hazard in terms that are useful to risk managers. The National Academy of Science recommended that risk assessment be considered "the characterization of the potential adverse health effects of human exposure to environmental hazards."[4] Risk assessments should represent an objective analysis of all the relevant information and should characterize the likelihood that a particular level of exposure to a given contaminant will produce a specific effect in humans or wildlife.

During the early years of the environmental movement (1960 to 1975), regulatory agencies often made decisions largely based on political pressures, social concern, and the availability of money or technology. This approach persisted for perhaps ten years until the late 1970's when it was recognized that there were too many chemicals and problems to attack in such a subjective and uneven manner.[5] Quantitative risk assessments were heralded as the solution to this problem. It was anticipated that such an objective analysis would help risk managers interpret and prioritize the implications of hundreds of toxicity studies. It became apparent that the risk assessment process assumed an important role in meeting society's need to establish an objective and standardized procedure to evaluate complex sets of scientific data.

2. *See e.g.*, Ames, *supra* note 1; Munro and Krewski, *Risk Assessment and Regulatory Decision Making*, 19 FD. COSMET. TOXICOL. 549 (1981); NATIONAL RESEARCH COUNCIL, RISK ASSESSMENT IN THE FEDERAL GOVERNMENT: MANAGING THE PROCESS (National Academy Press, 1983); Ames, *Dietary Carcinogens and Anticarcinogens*, 221 SCIENCE 1256 (1983); Ames, Magaw & Gold, *Ranking Possible Carcinogenic Hazards*, 236 SCIENCE 271 (1987); Office of Science and Technology Policy, *Chemical Carcinogens; A Review of the Science and its Associated Principles*, 50 Fed. Reg. 10,372 (1985); Office of Technology Assessment, Assessment of Technologies for Determining Cancer Risks From the Environment (June 1981).

3. *See* Paustenbach, *A Survey of Health Risk Assessment*, in THE RISK ASSESSMENT OF ENVIRONMENTAL AND HUMAN HEALTH HAZARDS: A TEXTBOOK OF CASE STUDIES 29 (D.J. Paustenbach ed. 1989).

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5. Ruckelshaus, *Science, Risk, and Public Policy*, 221 SCIENCE 1026, 1027 (1983).

One benefit of risk assessment is that the truly important problems can be identified and prioritized, which in turn helps risk managers make decisions that are reasonable and cost effective.

Risk assessments appeal to regulators and the courts because they assemble and interpret all the pertinent information.[6] Assessments appear more relevant to judges and juries than the results of single or multiple toxicity tests because the significance of the substances' physical properties, acute and chronic toxicity, metabolism, interspecies differences, environmental fate, degree of human exposure, and background concentrations are all considered.[7] A risk assessment benefits the non-scientist decisionmaker by discussing and interpreting the interactions of these many factors in an understandable manner.

Interestingly, health risk assessments are not an entirely recent activity. Various examples date back almost 3,000 years.[8] They have been used by regulatory agencies for at least thirty years, most notably within the U.S. Food and Drug Administration (FDA).[9] Many of our existing environmental and occupational health standards have been, at least in part, based on the results of low-dose extrapolation models and exposure assessments. In addition, the dose extrapolation models used today were originally developed in the 1960's by radiation biologists concerned with the cancer hazard posed by exposure to medical x-rays and nuclear fallout.[10] More recently, risk assessment methodologies have been used to set standards for chemical carcinogens including pesticide residues, drinking water guidelines, ambient air standards, as well as exposure limits for contaminants found in indoor air, the workplace, consumer products, and other settings.[11]

6. Preuss & Ehrlich, *The Environmental Protection Agency's Risk Assessment Guidelines*, 37 J. AIR POLL. CONTROL A. 784 (1987).

7. Paustenbach, *supra* note 3, at 29.

8. *Id.*, at 32-40.

9. *See, e.g.*, Rodricks, *Origins of Risk Assessment in Food Safety Decision Making* 7, J. AM. COLL. TOXICOL. 539 (1989); Lehmann and Fitzhugh, *100 Fold Margin of Safety*, 18 Q. BULL. A. FOOD & DRUG OFF. U.S. 33 (1954); LEHMANN, APPRAISAL OF THE SAFETY OF CHEMICALS IN FOODS, DRUGS AND COSMETICS (Assoc. of Food and Drug Officials of the United States, Topeka, KS., 1959).

10. *See* Rodricks, *Origins of Risk Assessment*, *supra* note 9.

11. *See* Paustenbach, *supra* note 3.

Beginning in about 1984, risk assessments began to be used in personal injury cases involving exposure to toxic chemicals. The objective was to estimate the possible level of human exposure prior to the onset of the alleged injury. When the exposure estimates were shown to be relatively precise and reasonable, they were instrumental in refuting or supporting medical opinions involving causation.

Risk assessments were welcomed by Congress, environmental groups, industry, and the public because these groups expected them to organize and interpret what appeared to be an unmanageable amount of information. It was hoped that risk assessments would provide an objective approach to identifying and prioritizing hazards, as well as, help determine causation in toxic tort litigation. Regrettably, something went wrong. Few scientists, including those employed by industry, have been completely satisfied with the way risk assessments have been conducted by government agencies or their contractors.

III. CRITICISMS OF HEALTH RISK ASSESSMENTS

Scientists, engineers and attorneys have identified a number of shortcomings with what has become a typical approach for regulatory agencies conducting health risk assessments. These criticisms involve all four portions of the risk assessment process: hazard identification, dose-response assessment, exposure assessment and risk characterization.[12] Perhaps the primary concern has been that the rigidity built into regulatory assessments, caused by pressure to repeatedly adopt conservative assumptions, often does not allow all of the scientific information to be considered.[13] One consequence of this rigidity is that these assessments predict health risks much higher than those which are likely to

12. *See* NATIONAL RESEARCH COUNCIL, *supra* note 2, at 21, for a figure outlining the elements of risk assessment and risk management.

13. *See, e.g.*, Paustenbach, Shu & Murray, *A Critical Examination of Assessments of the Health Risks Associated with TCDD in Soil*, 6 REG. TOXICOL. & PHARM. 284 (1986); Turnbull & Rodricks, *Assessment of Possible Carcinogenic Risk to Humans Resulting From Exposure to Di(2-ethylhexyl)phthlate (DEHP)*, 4 J. AM. C. TOXICOL. 111 (1985); Food Safety Council, *Quantitative Risk Assessment*, in FOOD SAFETY ASSESSMENT 137, 159 (1980); Park & Snee, *Quantitative Risk Assessment: State-of-the-Art for Carcinogenesis*, 37 AM. STATISTICIAN 427, 428 (1983); Maxim & Harrington, *A Review of the Food and Drug Administration Risk Analysis for Polychlorinated Biphenyls in Fish*, 4 REG. TOXICOL. & PHARM. 192 (1984); SIELKEN, *The Capabilities, Sensitivity, Pitfalls, and Future of Quantitative Risk Assessment*, in ENVIRONMENTAL HEALTH RISKS: ASSESSMENT AND MANANGEMENT 95 (R.S. McColl ed. 1987).

exist. Another problem is that such evaluations have often focused only on the maximally exposed individual (MEI) rather than the typical person. As a result, many assessments do not yield results which apply to the vast majority of people in the community; the primary concern of risk managers.

One criticism of risk assessment was recently raised by Dr. Barry Commoner, a well-known environmental spokesperson, at a gathering of EPA employees. He was reported to have said, "The environment will not be protected by the current practice of finding an acceptable level of harm from an environmental pollutant and then issuing rules allowing industry to pollute to that level."[14] Such a characterization of the risk assessment process is not accurate and is likely to prevent a useful approach from maturing into the scientific tool that is clearly needed by regulators and the public.

It is true that one use of risk assessment is to identify levels of emissions which would not pose a significant human or environmental risk, based upon the degree of human exposure, and the associated risk. The need for such analyses came about because regulators learned that it was theoretically impossible, as well as impractical and unnecessary, to reduce the emissions of all chemicals to zero or undetectable levels.[15] The plea that we must stop pollution at the source and not allow industry to pollute up to certain levels is too simplistic. One reason that we cannot eliminate "all" emissions is that we can now identify quantities as small as one part per quadrillion (ppq). With detection of such low levels possible, agencies would have to declare even the most healthy diet and the cleanest air to be potentially hazardous if exposure to measurable levels of carcinogens were deemed unacceptable. For example, the ambient air in the north woods of Maine contains detectable levels of polycyclic aromatics which are responsible for the pine odor, but which are carcinogenic in animals. Similarly, although perhaps it is in conflict with what the public has been told, naturally occurring carcinogens present in vegetables pose a cancer hazard perhaps 10,000 times greater than that posed by the pesticide residues in our diet.[16]

14. *EPA Critic Enters the Lion's Den and is Showered by Wild Applause*, N.Y. Times, Jan. 15, 1988, at B6, col. 4.

15. *See* Preuss & Ehrlich, *supra* note 6 (discussing the detailed and expansive risk assessment techniques developed at EPA).

16. Ames, *Dietary Carcinogens*, *supra* note 2, at 1258.

Although the elimination of exposure to all non-naturally occurring substances (xenobiotics) may seem a worthwhile objective, attempts to set regulations at such levels appear to be foolish, and would certainly not be the best use of America's limited resources. If there is any doubt about the finite quantity of financial resources available in the United States, and virtually every other country, one needs only to follow the current debate regarding the age criteria for deciding when to stop treating patients who could be cured and/or functional after medical treatment but are perhaps too old to justify the expense.[17]

Some public interest groups have taken the position that risk assessments allow too much variability in the implementation of legislation in an already "loose" regulatory environment. It has been claimed that setting acceptable risk levels for environmental emissions is inappropriate since persons can be placed at risk even though they receive no direct benefit. Although these are important issues, they all appear to be based on some level of misrepresentation or misunderstanding. The fact is that risk assessments should help standardize the way we regulate chemicals, thus reducing the arbitrariness which has sometimes been present. Furthermore, living in proximity to others, especially in a technological society, by definition, exposes some people to risks not of their own making.

Widespread acceptance of risk assessment will occur when there is better understanding of the process by all parties or when a better alternative is identified. Regulatory agencies cannot arbitrarily decide that it is acceptable for the public to be exposed to significant (*e.g.*, 10^{-3} or greater) levels of risk. For example, for environmental risks to be deemed acceptable by regulatory agencies, they usually need to be negligible or de minimis (*e.g.*, of such little importance as to be of no concern). Determining whether a risk is significant is influenced by a number of factors including the number of persons exposed, the likelihood and degree of exposure, and the certainty of the biologic data.[18] I am optimistic that, because chemists can now detect the presence of contaminants at concentrations less than one ppq, the public has come to

17. *Ethicist Draws Fire with Proposal for Limiting Health Care to Aged*, Wall St. J., Jan. 22, 1988, at 23, col. 1.

18. *See, e.g.*, Travis, Richter, Crouch, Wilson & Klema, *Cancer Risk Management*, 21 ENVTL. SCI. & TECH. 415, 419 (May 1987) (a review of federal regulatory decisions, which concludes that there is a consistency to the agencies' regulatory decisions).

recognize that measurable exposure to a chemical carcinogen or a reproductive toxicant does not necessarily mean the associated risk is unacceptable. If this were the case, people would not ingest alcohol, diet soda, coffee, tea, orange juice or mineral water since each contains measurable, albeit small, quantities of carcinogens.

Research of the past few years has been useful in identifying the levels of risks which the public finds acceptable. We have learned that the acceptability of a risk is a judgment that each person must reach: what is an acceptable risk to one person may be thoroughly unacceptable to another.[19] We have learned that most persons are comfortable with accepting certain levels of involuntary risks if they are very small. For example, risks in the vicinity of 1 in 1,000,000 (the chance of being struck and killed by lightning) seems to be acceptable to nearly all persons. As expected, the cost of reducing risks to such a level is not trivial. For this and other reasons, many environmental regulatory decisions allow involuntary risks to be as great as 1 in 10,000; especially if the number of exposed persons is small. The cost to lower the risk can be very high and the true risk may actually be far less than that predicted in a conservative risk analysis.[20]

My experience indicates that the strengths and weaknesses of each portion of a risk assessment need to be understood if an objective and fair resolution of environmental issues is to occur. The various elements of a risk assessment play a pivotal role in identifying appropriate clean-up levels and in helping to resolve personal injury cases in a fair manner.

IV. Pitfalls in Risk Assessment

The risk assessment process has four basic steps: hazard identification, dose-response assessment, exposure assessment and risk characterization.[21] In light of the economic impact that environmental regulations can have on a firm or the community, these pitfalls need to be recognized.

19. *See* W. Lowrance, Of Acceptable Risk 92 (1976).

20. *See* Travis, Richter, Crouch, Wilson & Klema, *supra* note 18, at 419; Rodricks, Brett & Wrenn, *Significant Risk Decisions in Federal Regulatory Agencies*, 7 Reg. Toxicol. and Pharm. 307, 315 (1987); Travis & Hattemer-Frey, *Determining Acceptable Levels of Risk*, 22 Envtl. Sci. & Tech. 873, 875 (1988).

21. *See* National Research Council, *supra* note 12.

FIGURE 1

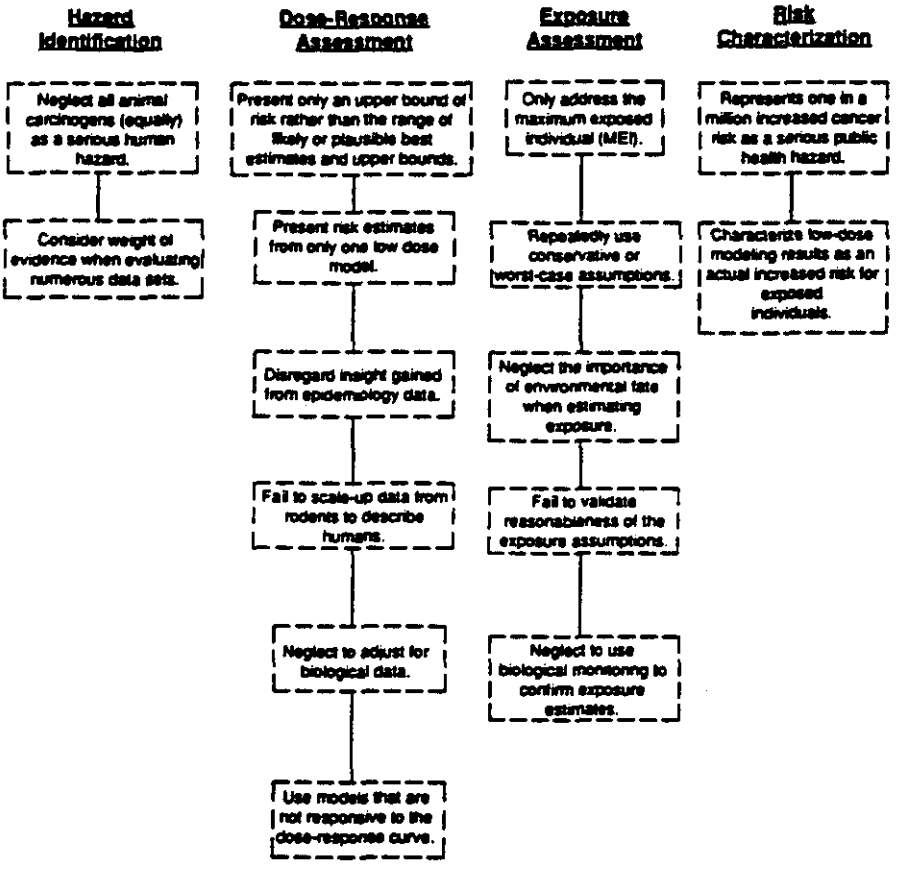


FIGURE 1: Examples of possible pitfalls in conducting or presenting health risk assessments.

A. *Hazard Identification*

In the hazard identification step of the risk assessment, there has been a tendency to consider all animal carcinogens as posing an equally serious human health hazard. In fact, carcinogens vary dramatically in their carcinogenic and/or mutagenic potency. Specifically, a weak carcinogen may require a dose 10,000,000 fold greater than that of a potent carcinogen to produce the same degree of carcinogenic response.[22] In addition, the susceptibility between species, the slope of the dose-response curve for the various toxic endpoints, pharmacokinetics, the epidemiological experience and the mechanism of action all need to be considered when attempting to predict the potency of a chemical in humans.

22. Ames, *Dietary Carcinogens supra* note 2, at 1261.

The importance of the above factors cannot be overstated since they may well explain why six hundred chemicals have been found to produce tumors in animal studies yet less than twenty are known to be human carcinogens.[23] Even after accounting for the statistical shortcomings of most epidemiological studies, it is clear that not all carcinogens pose an equivalent human hazard. The same can be said of developmental and reproductive toxicants. The challenge is to determine to what degree we need to limit exposure to each of these toxicants to insure that the risk to humans is negligible.[24]

The criteria by which the risk assessor determines that a chemical poses a significant carcinogenic or developmental threat to humans involves consideration of at least six factors.[25] For carcinogens, most of the important parameters have been identified and discussed in numerous published papers.[26] At least the following parameters should be considered when determining that an animal carcinogen may pose a human cancer hazard: number of animal species affected, the number and types of tumors occur-

23. Ames, *supra* note 1.

24. *See e.g.*, Rodricks, Brett & Wrenn, *supra* note 20, at 315; Travis & Hattemer-Frey, *supra* note 20, at 875-76; L. LAVE, ED. QUANTITATIVE RISK ASSESSMENT IN REGULATION (The Brookings Institute 1982) 153 (using estimation of lung cancer deaths caused by coke oven emissions as an example); Nichols & Zeckhauser, *The Perils of Prudence: How Conservative Risk Assessments Distort Regulation*, 8 REG. TOXICOL. & PHARM. 61 (1988) (arguing that current conservative assessment techniques leads to unnecessary overestimation of risks).

25. Critical factors in the hazard identification of chemical carcinogens and developmental toxicants:

CARCINOGENS	DEVELOPMENTAL TOXICANTS
- Number of different species affected	- Number of animal speciel affected
- Number of different types of neoplasms in one or more species	- Difference between species
- Spontaneous incidence in appropriate control group neoplasms induced in treated groups	- Relevancy of route of administration
- Dose-response relationship	- Multiplicity and nature of developmental effects among litters
- Malignancy of induced neoplasms	- Number of litters or fetuses being affected
- Genotoxicity, measured in an appropriate battery of tests	- Rare vs. common malformations
	- Ratio of adult and developmental NOEL or LOEL

Adapted from Squire, *Ranking Animal Carcinogens: A Proposed Regulatory Approach*, 214 SCIENCE 877, 878 (1981); Johnson, Christian, Dansky & Gabel, *Use of the Adult Developmental Relationship in Prescreening for Developmental Hazards*, 7 TERATOGENESIS, CARCINOGENESIS, & MUTAGENESIS 273 (1987); Wang & Schwetz, *An Evaluation System for Ranking Chemicals with Teratogenic Potential*, 7 TERATOGENESIS, CARCINOGENESIS & MUTAGENESIS 133, 134 (1987).

26. *See, e.g.*, Munro & Krewski, *supra* note 2; Paustenbach, *supra* note 3; Sielken, *supra* note 13.

ring in the animals, the dose (relative to the acute toxic dose) at which the animals are affected, the dose/response relationship, and the genotoxicity of the chemical.[27] For the developmental toxicants, guidance has been provided by a number of researchers.[28] The primary factors are similar to those for carcinogens and include: the number of species affected, severity of the effect and the relationship of the dose which affects the mother compared to that which affects the offspring.[29]

During the past few years, regulatory agencies often placed an emphasis on any piece of data that supported the fact that a chemical posed a carcinogenic or developmental hazard, and little weight on data that suggested the chemical failed to cause these problems. Extraordinary confidence was placed on studies which indicated that a chemical may pose a particular hazard, irrespective of the study's quality. This approach was considered prudent and health protective. Recently, the scientific community and most regulatory agencies have come to recognize that not all data are equal, and that only data of similar quality should be judged equally. We have also learned through experience that it should not be necessary to spend huge sums of money to repeatedly conduct high quality toxicity studies simply to refute one or two poorly controlled ones. Further, when the conclusions reached from high quality data are overwhelming, spurious data must be de-emphasized or discarded. This philosophy, known as a "weight of evidence" approach, has been applied primarily to the hazard identification segment of risk assessment, but is also applicable to the exposure and dose-response assessment segments.[30]

27. Squire, *Ranking Animal Carcinogens: A Proposed Regulatory Approach*, 214 SCIENCE 877-78 (1981); *See also* EPA, *Guidelines for Carcinogenic Risk Assessment*, 51 Fed. Reg. 33,992, 34,000 (1986); California Department of Health Services, Guidelines for Carcinogenic Risk Assessment and Their Scientific Rationale A-12 - A-14 (1986).

28. *See, e.g.*, Johnson, Christian, Dansky & Gabel, *Use of the Adult Developmental Relationship in Prescreening for Developmental Hazards*, 7 TERATOGENESIS, CARCINOGENESIS, & MUTAGENESIS 273 (1987); Kimmel & Gaylor, *Issues in Qualitative and Quantitative Risk Analysis for Developmental Toxicology*, 8 RISK ANALYSIS 15 (1988); Johnson, *Cross-Species Extrapolations and the Biologic Basis for Safety Factor Determinations in Developmental Toxicology*, 8 REG. TOXICOL. & PHARM. 22 (1988); Wang & Schwetz, *An Evaluation System for Ranking Chemicals with Teratogenic Potential*, 7 TERATOGENESIS, CARCINOGENESIS & MUTAGENESIS 133 (1987).

29. Wang & Schwetz, *supra* note 28, at 135.

30. EPA Dioxin Task Force, A Cancer Risk-Specific Dose Estimate for 2, 3, 7, 8-TCDD 3 (1987) (External Review Draft).

The following statements from the 1986 EPA Cancer Guidelines[31] summarize their approach to applying the weight of evidence test:

> The overall scheme for categorization of the weight of evidence of carcinogenicity of a chemical for humans uses a three-step process. (1) The weight of evidence in human studies or animal studies is summarized; (2) these lines of information are combined to yield a tentative assignment to a category; and (3) all relevant supportive information is evaluated. Relevant factors to be included along with the tumor information from human and animal studies include structure-activity relationships; short-term test findings; results of appropriate physiological, biochemical, and toxicological observations; and comparative metabolism and pharmacokinetic studies. The nature of these findings may cause one to adjust the overall categorization of the weight of evidence.

This scheme is a good first attempt at bringing more reason to the hazard identification process. One advantage of the weight of evidence approach is that when new information is available, it is considered and weighed fairly against the old.

B. *Dose-Response Assessment*

Perhaps the most uncertain portion of assessments of chemical carcinogens is the low-dose extrapolation assessment. For this reason, it offers a plethora of opportunities for technical improvement and for better communication of the uncertainties to the risk manager. At best, science has a limited ability to use the results of standard rodent bioassays to understand the human cancer hazard posed by typical levels of exposure.[32] The main reason is that we do not yet fully understand all of the various possible mechanisms of action for carcinogens. Accordingly, we must rely on a model or theory to estimate the human response to environmental pollutants since they are generally exposed to doses at least one-thousand-fold below the lowest animal dose

31. EPA Guidelines, *supra* note 27, at 34,000.

32. Ames, *Dietary Carcinogens supra* note 2, at 1261; *See also* Anderson, *Quantitative Risk Assessment and Occupational Carcinogens*, 3 APPL. IND. HYG. 267, 268 (1988); Conolly, Reitz, Clewell & Andersen, *Biologically Structured Models and Computer Simulation: Application to Chemical Carcinogenesis*, 2 COMMENTS TOXICOL. 305 (1988); Crump, *An Improved Procedure for Low-Dose Carcinogenic Risk Assessment from Animal Data*, 5(5) J. ENVTL. PATH. TOX. 339 (1984); Crump & Howe, *The Multistage Model with a Time-Dependent Dose Pattern: Applications to Carcinogenic Risk Assessment*, 4 RISK ANALYSIS 163 (1984) (all of these articles present and discuss models to extrapolate animal data to the human situation).

tested.[33] Although rarely accounted for in the dose-response models, such doses may well be easily handled by the protective biologic mechanisms in humans.[34]

The reason for conducting a dose-response assessment is to understand what response might occur, if any, one-hundred to one-thousand-fold below the lowest dose tested in rodents (since these are the levels to which humans are typically exposed). Because it would require the testing of thousands of animals to observe a response at such low doses, mathematical models are used to predict the response. To understand the level of uncertainty in the dose extrapolation process and the typical regulatory use of low-dose models, the dose-response curve must be understood. In the example shown in Figure 2, 100% of the animals responded at a dose of 100 milligrams per kilogram per day (mg/kg-day), 50% responded at 50 mg/kg-day, and 5% of the animals developed the response at 5 mg/kg-day. None of the animals were affected at a dose of 1 mg/kg-day, and this is called the no observed adverse effect level (NOEL). Therefore, 5 mg/kg-day constituted the lowest observed effect level (LOEL). As shown, the experimental data range over only a factor of 100; between 1 and 100 mg/kg-day. The challenge, which can contain a high degree of uncertainty, is to estimate what might occur (if anything) in humans exposed to doses perhaps as low as 0.001 mg/kg-day.

There are at least six serious pitfalls into which scientists can slip during the conduct of a dose-response assessment (Figure 1). The first pitfall is to present only the upper-bound risk from the cancer models rather than identifying the range of likely or best estimates, as well as the upper bounds of the risk. The objective of the bounding techniques is to attempt to account for the statistical uncertainty in the results of the animal tests. However, the degree of potential conservatism of the bounding procedure and the fact that zero risk is as likely as the upper-bound value of risk is rarely reported in risk characterizations. The result is that the risk manager rarely is fully aware of the breadth of equally plausible risk estimates. For example, the cancer risk associated with

33. Ames, *supra* note 1, at 382 ("[E]xtrapolating linearly from the enormous doses of rat tests to low-dose human exposure may be much too pessimistic even for those carcinogens which are mutagens.")

34. *Id.*, at 381; BUS & GIBSON, *Body Defense Mechanisms to Toxicant Exposure*, in 3B PATTY'S INDUSTRIAL HYGIENE AND TOXICOLOGY 143 (J. Lewis and L. Cralley eds. 1985).

FIGURE 2

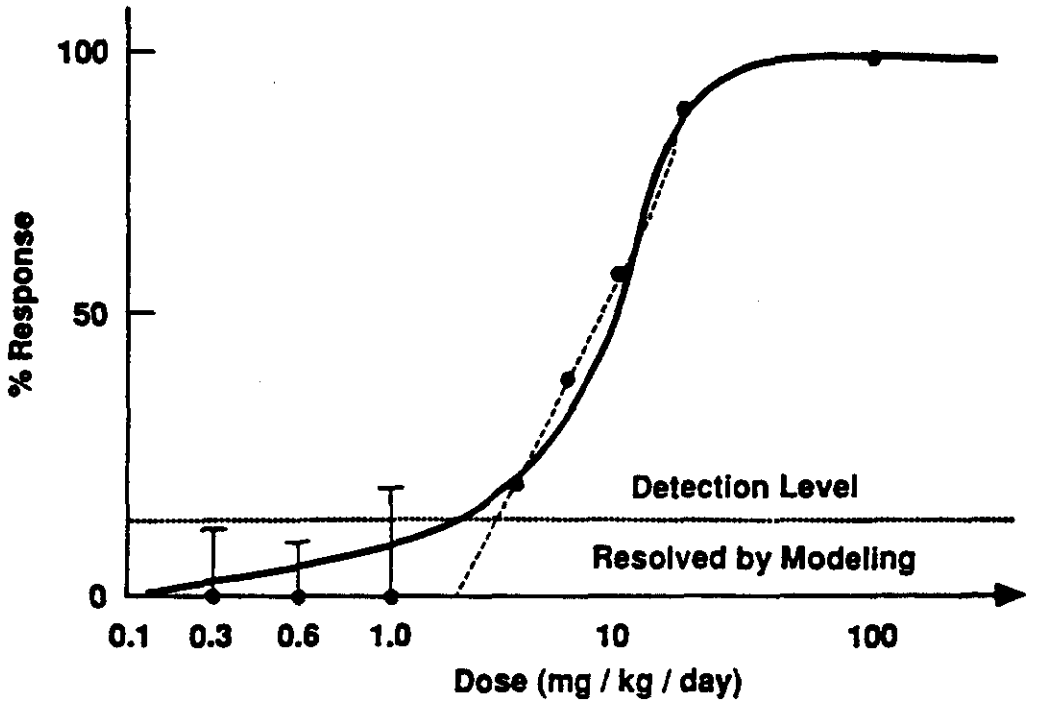


FIGURE 2: A dose-response curve from a carcinogenicity study. The solid line is a best fit of the eight data points identified in the test. The three lowest data points indicate that at these doses, no increased incidence in tumors was observed in the test animals. The error bars on the three lowest doses indicate the statistical uncertainty in the test results since a limited number of animals were tested (n = 100). In an effort to derive risk estimates that are unlikely to underestimate the risk, the models usually derive risk estimates based on the estimated upper bound of the response, rather than the best estimate.

exposure to chloroform in drinking water has been reported to be as high as one in ten thousand using the upper bound estimate of the multi-stage model. However, using the same model, the best or maximum likelihood estimate of the risk is about one in a million and the lower bound estimate is zero. Therefore, the plausible range of risk is as high as one in ten thousand and as low as zero. When biological factors are considered, such as its lack of genotoxicity, the carcinogenic risk associated with the levels of chloroform in chlorinated drinking water is most likely to be negligible.[35]

Reliance on the results of only one mathematical model is the second potential pitfall in the dose-response assessment. To the

35. REITZ, QUAST, STOTT, WATANABE & GEHRING, *Pharmacokinetics and Macromolecular Effects of Chloroform in Rats and Mice: Implications for Carcinogenic Risk Estimation*, 1980 WATER CHLORINATION 983, 991.

surprise of many scientists and attorneys, there are at least six different modeling approaches that may need to be considered when estimating the risks at low doses. These models include the probit, multihit, multistage, Weibull, one-hit, and, when possible, the Moolgavkar - Knudson - Venzon (MKV) biologic-based approach. Nearly all of them can yield results which are plausible.[36] Although it has been claimed that models which lack low dose linearity are not appropriate for carcinogens, the scientific support for this assertion is not compelling, especially for chemicals which are not genotoxic. Except for those chemicals which are known to be initiators or mutagens, no single statistical model can be expected to accurately predict the low-dose response with greater certainty than another.[37] One approach is to present the best estimate of the risk from the two or three models which are considered equally reasonable, as well as, the upper and lower-bound estimates from those models. The estimates should be accompanied by a rationale as to why one model appears more reasonable for that particular chemical or data set. The model's responsiveness to the data or the most likely response due to biologic considerations should be the criteria for selection.

Adoption of this approach would give decisionmakers the benefit of access to pertinent data and an understanding of the uncertainty in the results. Sielken has described how such an approach might be implemented and has identified criteria for conducting a dose-response assessment.[38] If there is a biological or statistical reason to favor one model over another, then the weight of evidence approach should be used to select the most justifiable value. Such an approach was recently attempted by the EPA in its reevaluation of dioxin (TCDD).[39] The diversity of views between various regulatory agencies and scientists within the United States and in other countries on safe levels of exposure to TCDD is illustrated in Figure 3.

Some generalizations can be made about low-dose models. It is noteworthy that the various models will usually fit the rodent data

36. FOOD SAFETY COUNCIL, *supra* note 13, at 159; Park & Snee, *supra* note 13, at 428.

37. *See, e.g.*, Turnbull & Rodricks, *supra* note 13; Maxim & Harrington, *supra* note 13; Sielken, *supra* note 13, at 105.

38. Sielken, *Some Issues in the Quantitative Modeling Portion of Cancer Risk Assessment*, 5 REG. TOXICOL. & PHARM. 175 (1985) (listing and discussing 20 criteria for evaluating the dose response extrapolation in a cancer bioassay)

39. See *supra*, note 30.

FIGURE 3

Weight of Evidence Evaluation
Dioxin (1988)
(upper bound dose at 1 in a million risk)

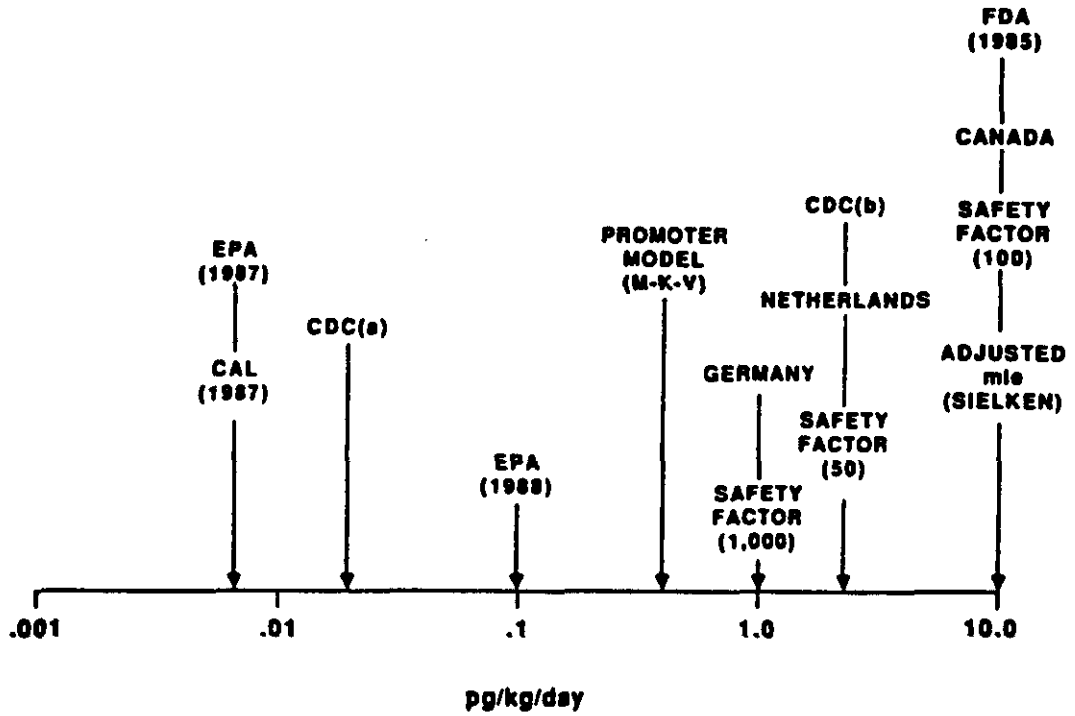


FIGURE 3: Application of the weight of evidence approach should improve each phase of the risk assessment process. Recently, the U.S. EPA evaluated all the various national and international health guidelines for dioxin in an effort to select the most appropriate one to use in the coming years in the United States. As shown here, equally creditable scientific bodies can occasionally have very different views about what constitutes a safe level of human exposure to a chemical. Adapted from ENVIRONMENTAL PROTECTION AGENCY, A CANCER RISK-SPECIFIC DOSE ESTIMATE FOR 2,3,7,8-TCDD (1988) (Draft), at 4.

in the observable dose region, but that they vary in the unobserved, but all important, low-dose region (Figure 4). It should also be recognized that the results of the six most commonly used low-dose models usually vary in a predictable manner.[40] In general, although not in all cases, the one hit and linearized

40. The results of low-dose extrapolation models usually vary in the following predictable manner:

MODEL	PREDICTED RISK
Linear	highest
One-Hit	↑
Multistage	|
Weibull	|
M.K.V.	|
Multi-Hit	|
Logit	|
Probit	lowest

FIGURE 4

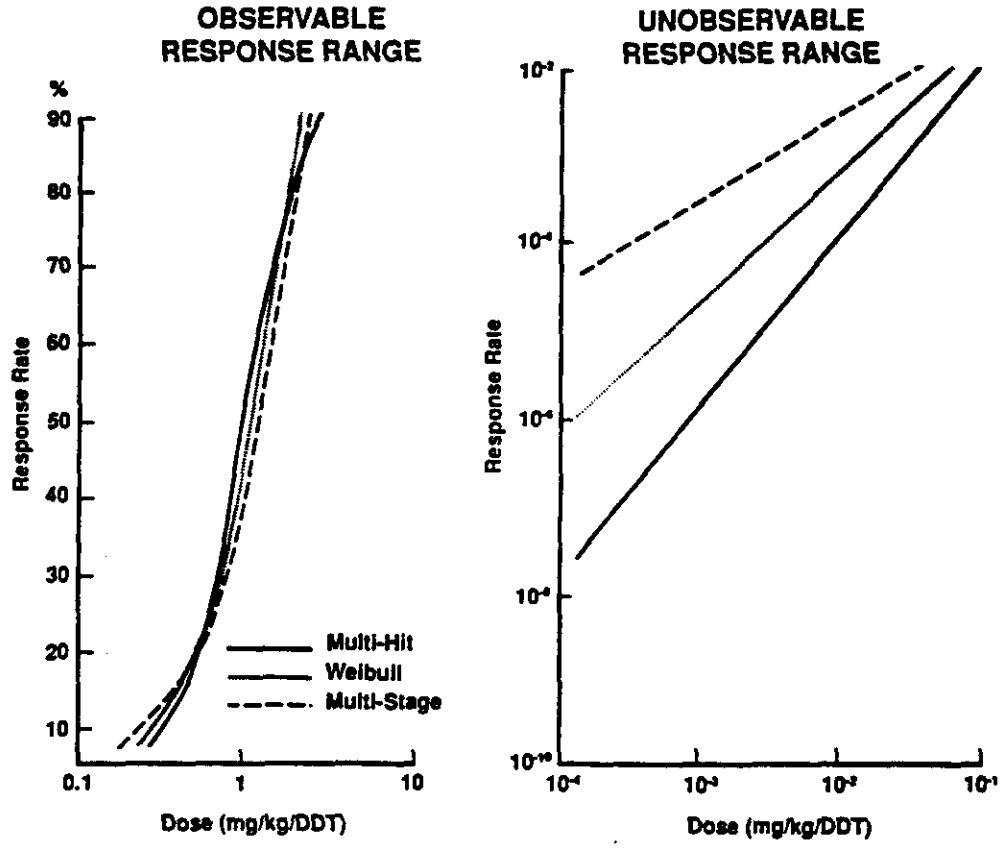


FIGURE 4: The fit of most dose-response models to data in the observable range is generally similar. However, due to the differences in the assumptions upon which the equations are based, the risk estimates at low doses can vary dramatically between the different models.

multi-stage models will predict the highest risk and the probit model will predict the lowest.[41] The results vary in a predictable manner because the models are based on different mathematical equations which are expected to describe the chemical's likely behavior in the low dose region.

Over the past fifteen years, mathematicians and toxicologists have not been able to present a compelling reason to choose one extrapolation model over another, so regulatory agencies arbitrarily adopted the one that usually predicted the highest risk *i.e.*, the linearized multi-stage model (to insure that they were above accusation that they were not protective of the public health). The statistical underpinnings of the multi-stage model, the one

41. Munro & Krewski, *supra* note 2, at 554.

most widely accepted, are the best documented of the various models. However, like the other statistical models it can not make use of most of the biologic information on a substance. Hopefully, toxicologists now know enough about the likely mechanism of carcinogenicity of enough chemicals to provide sufficient insight to select the most appropriate form of the multistage model, or several different models or approaches, to identify an acceptable level of human exposure.[42] For example, a substantial number of scientists (although certainly not all of them) believe that there are at least three mechanisms by which chemicals may produce a carcinogenic response: repeated cytotoxicity, promotion, and initiation. Butterworth has suggested that at least eight different classes of carcinogens exist.[43] These distinctions are important since the appropriate model for estimating the cancer risk for humans exposed to low doses of a cytotoxicant or promotor should be markedly different than that for an initiator.[44]

In general, the scientific underpinnings of the dose-response models are based on our understanding of the cancer process caused by exposure to ionizing radiation and chemicals that are initiators.[45] Both types of agents may well have a nearly linear or a linear response in the low dose region. However, promoters and cytotoxicants need not have a linear dose response curve. Scientific data increasingly suggest that they would be expected to be very non-linear at low doses and, more importantly, probably have a genuine or practical threshold (a dose below which no response [risk] would be present).[46] The increased acceptance of this postulate is evidenced by EPA's recent position that the linearized multi-stage model is inappropriate for dioxin, thyroid type carcinogens, NTA, and, presumably, similar non-genotoxic

42. *Id*, at 556; BUTTERWORTH & SLAGA, NONGENOTOXIC MECHANISMS IN CARCINOGENESIS (1987); WEISBERGER & WILLIAMS, *Chemical Carcinogens*, in CASARETT AND DOULL'S TOXICOLOGY 84, 134 (Doull, Klaassen & Amdur eds. 3d ed. 1986). *See also* Butterworth, *Nongenotoxic Carcinogens*, 7 CIIT ACTIVITIES 1 (Dec. 1987).

43. BUTTERWORTH & SLAGA, *supra* note 42.

44. *Id.*; Weisburger & Williams, *supra* note 42, at 134; Anderson, Clewell, Gargas, Smith & Reitz, *Physiologically Based Pharmacokinetics and the Risk Assessment Process for Methylene Chloride*, 87 TOXICOL. & APP. PHARM. 185 (1987).

45. E.J. CALABRESE, PRINCIPLES OF ANIMAL EXTRAPOLATION 518-20 (1983); NATIONAL RESEARCH COUNCIL, THE EFFECTS ON POPULATION OF EXPOSURE TO LOW LEVELS OF IONIZING RADIATION 21-23 (National Academy Press 1980).

46. Squire, *supra* note 27; Paynter, Burin, Jaeger & Gregorio, *Goitrogens and Thyroid Follicular Cell Neoplasia: Evidence for a Threshold Process*, 8 REG. TOXICOL. PHARM. 102 (1988) (commenting on research indicating a threshold for thyroid follicular neoplasia).

chemicals.[47] For these types of chemicals, a threshold model, the MKV model or one of the other biologically-based models appears to be more appropriate.[48] The extrapolation process is improved further if a physiologically-based pharmacokinetic model (PB-PK) has also been used to correctly calculate the delivered dose and scale-up the rodent data to humans.[49]

The third pitfall in the dose-response assessment is to disregard the insight gained from epidemiological data. Traditionally, it has been claimed that epidemiologic studies are almost never as statistically robust as the animal studies and, therefore, are not very useful.[50] Acceptance of this assertion seems inappropriate because epidemiological studies can establish the degree of confidence that should be placed in the results of low-dose extrapolation models.[51] For example, in 1982 it was claimed that workers exposed for 8 hrs/day for 40 years to the OSHA standard for ethylene dibromide (20 ppm) incurred a risk of 999 in 1,000 of developing cancer due exclusively to this level of occupational exposure.[52] However, epidemiological studies of the actual can-

47. EPA, *supra* note 30, at 3; ANDERSON & ALDEN, *Risk Assessment for Nitrilotriacetic Acid (NTA)*, in THE RISK ASSESSMENT OF ENVIRONMENTAL AND HUMAN HEALTH HAZARDS: A TEXTBOOK OF CASE STUDIES 390, 422 (D.J. Paustenbach ed. 1989); Paytner, *supra* note 46.

48. *See, e.g.*, Krewski, Brown & Murdoch, *Determining "Safe" Levels of Exposure: Safety Factors or Mathematical Models?* 4 FUND. APP. TOXICOL. S383, S391-2 (1984); Moolgavkar, *The Multistage Theory of Carcinogenesis and the Age Distribution of Cancer in Man*, 61 J. NAT'L CANCER INST. 49 (1978); Moolgavkar & Venzon, *Two-Event Models for Carcinogenesis: Incidence Curves for Childhood and Adult Tumors*, 47 MATH. BIOSCIENCES 55 (1979); Ellwein & Cohen, *A Cellular Dynamics Model of Experimental Bladder Cancer: Analysis of the Effect of Sodium Saccharin in the Rat*, 8 RISK ANALYSIS 215 (1988); Moolgavkar, Dewanji & Venzon, *A Stochastic Two-Stage Model for Cancer Risk Assessment: The Hazard Function and the Probability of Tumor*, 8 RISK ANALYSIS 383 (1988).

49. Andersen, *Incorporating Pharmacokinetics and Risk Assessment Into the Setting of Occupational Exposure Limits: The Stodinger Lecture*, 3 *Appl. Ind. Hyg.* 10 (1988); Andersen, Clewell, Gargas, Smith & Reitz, *supra* note 44; D'Souza & Boxenbaum, *Physiological Pharmacokinetic Models: Some Aspects of Theory, Practice and Potential*, 4 TOXICOL. INDUS. HEALTH 151 (1988).

50. Office of Science and Technology Policy, *supra* note 2, 10,375, 10,421; LAYARD & SILVERS, *Epidemiology in Environmental Risk Assessment*, in THE RISK ASSESSMENT OF HUMAN AND ENVIRONMENTAL HEALTH HAZARDS: A TEXTBOOK OF CASE STUDIES 157, 160 (D.J. Paustenbach, ed. 1989); *Dioxin Risk to Humans is Minimal*, Pesticide & Toxic Chemical News, Oct. 26, 1988, at 24.

51. LAYARD & SILVERS, *supra* note 50, at 160; Pesticide & Toxic Chemical News, *supra* note 50 (providing four suggestions by Dr. Vernon N. Houk of the Center for Disease Control, "[c]oncerning epidemiology that would be useful" for risk assessment); See also Dinman & Sussman, *Uncertainty, Risk, and the Role of Epidemiology in Public Policy Development*, 25 J. OCCUP. MED. 511, 514-5 (July 1983) (a test of a Proposed Epidemiologic Study Scoring Method)

52. *Adequate Standards or Cancellation, Rep. Miller Says of EDB*, Pesticide & Toxic Chemical News, July 13, 1983, at 26.

cer incidence in workers did not show an increase in the cancer rate even though they had been exposed to concentrations as high as 20 ppm for about fifteen to twenty-five years.[53] When epidemiological data are available, it seems scientifically inappropriate to blindly accept the results of mathematical models which analyze only rodent data without giving serious consideration to the human experience.[54] At the very least, epidemiological data can help bracket the range of reasonable risks associated with certain levels of exposure.[55] This "reality check" should be a part of every risk assessment, whenever possible.

Many scientists and regulators seem to have forgotten that virtually all published risk estimates for carcinogens and developmental toxicants are based on data collected in rodents which are often given doses 100 to 10,000 times greater than that to which humans are typically exposed.[56] Few people will argue that such testing is inappropriate or unnecessary for identifying potential carcinogens, but these data must be carefully interpreted before the risk to humans exposed at low doses can be estimated.[57] Among other things, it should be remembered that the rodent studies now used to predict human risk were never intended for that purpose.[58] These studies were designed to qualitatively identify potential human hazards, not to quantitatively estimate the human risk at low levels of exposure.[59]

Pitfall number four in dose-response assessment is the failure to carefully scale-up data from rodents to describe the human response. For purposes of risk assessment, statisticians and biolo-

53. Ames, *supra* note 1, at 380; Hertz-Picciotto, Gravitz & Neutra, *How Do Cancer Risks Predicted From Animal Bioassays Compare with the Epidemiologic Evidence? The Case of Ethylene Dibromide*, 8 RISK ANALYSIS 205 (1988).

54. LAYARD & SILVERS, *supra* note 50, at 160; Pesticide & Toxic Chemical News, *supra* note 50.

55. LAYARD & SILVERS, *supra* note 50 ("[E]pidemiology can play an important role in bracketing the risk estimates derived from animal experiments.")

56. Havendar, *Peanut Butter Sandwich Deadlier Than Muffins Containing EDB*, Wall St. J., April 4, 1984, at B11, col. 1. ("According to EPA's estimates, the average person consumes 5 to 10 micrograms of EDB a day...[t]hat quantity is less than a quarter-millionth of what, on a body-weight basis, the rats were given.")

57. *See, e.g.*, Sielken, *Quantitative Cancer Risk Assessments for 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD)*, 25 FOOD CHEM. TOXIC. 257 (1987).

58. *Id.*

59. S.L. FRIESS, *Risk Assessment: Historical Perspectives*, in PHARMACOKINETICS IN RISK ASSESSMENT, 8 DRINKING WATER AND HEALTH 3 (National Academy Press 1987); E. EFRON, THE APOCALYPTICS. CANCER AND THE BIG LIE, 308 (1984); Barr, *Design and Interpretation of Bioassays for Carcinogenicity*, 7 REG. TOXICOL. & PHARM. 422, 423 (1987).

gists have generally assumed that at a given dose (mg/kg-day) the rodent response to a chemical will be nearly identical to the human response; even though most scientists recognize that this will often not be true. This all-important assumption is no longer necessary, and risk assessors should move aggressively to incorporate a more scientifically defensible approach. Many factors need to be considered when trying to predict how humans will respond compared to rodents.[60] First, the biologic half-life between rodents and humans can be expected to vary for virtually all chemicals.[61] Often, for a given chemical, these differences will vary in a predictable manner based simply on the body weight to surface area ratio and/or life span.[62] As a result, regulators have used correction factors based on surface area in an attempt to adjust for the pharmacokinetic differences between rodents and humans. However, due to its simplicity, the surface area per body weight approach will frequently not account for the difference in half-life; additionally, the need for a correction factor depends on whether the carcinogen is the parent chemical or a metabolite.[63] Rather than rely on simple scale-up factors, we now have the capacity to accurately adjust our risk estimates to account for these differences by using physiologically-based pharmacokinetic (PB-PK) models.[64] They represent a mathematical approach to account for the various physiological and metabolic differences between the test species and humans including body weight, metabolic capacity and products, respiration rate, blood flow, fat content, and a number of other parameters.[65] The potential benefits of this approach have been so impressive that a special sym-

60. EPA, *supra* note 27, at 33,993-34,000; Bus & Gibson, *supra* note 34; Sielken, *supra* note 37; Whittemore, Grosser, & Silvers, *Pharmacokinetics in Low Dose Extrapolation Using Animal Cancer Data*, 7 FUND. & APP. TOXICOL. 183 (1986).

61. Biological half-life of selected chemicals (days):

SUBSTANCE	MOUSE	RAT	HAMSTER	GUINEA PIG	MONKEY	HUMAN
Carbon Tetrachloride	0.10	0.25	0.35	-	0.46	0.50
Dioxin	15.0	31.0	15.0	31.0	455.00	2800.00

62. Whittemore, Grosser & Silvers, *supra* note 60; Ramsey & Anderson, *A Physiologically Based Description of the Inhalation Pharmacokinetics of Styrene in Rats and Humans*, 73 TOXICOL. & APP. PHARM. 159 (1984).

63. D'Souza & Boxenbaum, *supra* note 49; HART & FISHBEIN, *Interspecies Extrapolation of Drug and Genetic Toxicity Data*, in TOXICOLOGICAL RISK ASSESSMENT 3 (Clayson, Krewski & Munro eds. 1985).

64. D'Souza & Boxenbaum, *supra* note 49.

65. *Id.*

posia was held by the National Academy of Sciences to discuss PB-PK models and encourage their use.[66]

The fifth, and possibly most important pitfall is failure to alter the risk estimates by considering biological information such as the time it takes for a tumor to appear, metabolic differences between species, and whether the chemical is genotoxic.[67] Generally, irrespective of the type of carcinogenic response, regulatory agencies will use a single curve fitting procedure to estimate the human risk. The result is usually based on three data points from a two-year rodent study.[68] The shortcomings associated with ignoring biological information are numerous. For example, nitrilotriacetic acid (NTA) produced kidney tumors in rodents, but only following very high doses. It was ultimately shown that at high doses, NTA produced chronic progressive nephrosis (CPN) due to repeated cytotoxity. The repeated toxic effects produced sufficient irritation to form bladder tumors.[69] However, at doses to which humans might be exposed, the tumors would not be expected to form. After a good deal of study, it was agreed that although the cancer models predicted a significant risk at low doses, no human risk was likely at the anticipated level of exposure.[70] This is one of many examples which illustrates that no matter how well the animal dose-response data are statistically analyzed, it is a serious pitfall to predict human health risks from rodent data without considering all the relevant biological data.

It is increasingly clear that numerous mechanisms are at work in the multi-step process of chemical carcinogenesis. Heretofore, we have divided chemical carcinogens into two broad classes: genotoxicants and nongenotoxicants. At one time, it was believed that all carcinogens were genotoxicants, chemicals which directly alter the DNA. Some believe that genotoxicants may act through point mutations, insertions, deletions, or changes in chromosome structure or number. These can be measured as chemical reactivity with the DNA, mutagenesis, induction of DNA repair, or cyto-

66. KREWSKI, MURDOCH & WITHEY, *The Application of Pharmacokinetic Data in Carcinogenic Risk Assessment*, in PHARMACOKINETICS IN RISK ASSESSMENT, 8 DRINKING WATER & HEALTH 441, 442 (National Academy Press 1987) (this volume was a result of the National Academy of Sciences symposia).

67. Sielken, *supra* note 38; Butterworth & Slaga, *supra* note 42.

68. Krewski, Brown & Murdoch, *supra* note 48.

69. *Id.*; Butterworth, *Nongenotoxic Carcinogens*, 7 CHEM. INDUS. INST. OF TOXICOL. ACTIVITIES 2 (1987).

70. ANDERSON & ALDEN, *supra* note 47.

genetic effects in bacterial or mammalian cell culture assays as well as in the whole animal. Conversely, nongenotoxic chemicals are those that lack genotoxicity as a primary biological activity.[71] While these agents may secondarily yield genotoxic events as a result of toxicity, such as hyperplasia (excessive cellular growth), their primary action does not involve reactivity with the DNA. Because at low doses nongenotoxicants may not produce toxicity, the primary reason for excessive cell turnover, many scientists expect them to possess a threshold dose below which no cancer hazard would be present. This is in contrast with genotoxicants which may have some risk, albeit small, even at very low doses.[72]

Pitfall six is the use of models which do not or are not capable of responding to the dose-response curve. As discussed by Sielken,[73] it does not seem appropriate to use models which are minimally responsive to the very costly information collected in standard lifetime rodent studies. By considering only one low-dose model, or by conducting only one statistical test for selecting the form of the model, we limit our ability to learn from the rodent data. One way to avoid this shortcoming is to conduct simulations of the model's responsiveness to alternative, but similar, data sets to insure that the extrapolation is reasonable. Some regulatory agencies, however, believe that too little is known about what might happen at low doses to change to less conservative approaches.[74]

What is meant by the phrase "responsive to the data?" Two terms are frequently used in this regard: fragile and insensitive. Fragile usually means that the model over-responds to the data while insensitive means that the risk estimates vary little irrespective of the rodent's response. The following example should illustrate the potential problem. Assume that two identical animal studies were conducted: one in New York and one in San Francisco. In each lab, there are one hundred test animals (fifty per sex were exposed to two doses and a control). At the conclusion, there was no increased tumor incidence in the females at any dose in either lab. However, in the males, we find that one additional

71. Butterworth, *supra* note 69.

72. *Id.*

73. Sielken, *supra* note 13; Sielken, *A Response to Crump's Evaluation of Sielken's Dose-Response Assessment of TCDD*, 26 FOOD CHEM. TOXICOL. 80 (1988).

74. California Department of Health Services, *supra* note 27; Crump, *A Critical Evaluation of a Dose-Response Assessment for TCDD*, 26 FOOD CHEM. TOXICOL. 79 (1988).

rat in San Francisco, at the 3 mg/kg-day dose, has a tumor compared to the test group in New York.[75] The controls had no increased incidence of this tumor type. To scientists, the biological difference between these results is insignificant; that is, the results are equivalent. To estimate the risk of having this chemical in our diet at a dose one thousand fold below the lowest dose tested in rodents, a model needs to be used.

Applying the multistage model, the one most frequently used in the United States, we find a significant difference in the maximum likelihood estimates (MLE) due only to the difference of one rat between the two studies. For example, as shown in our hypothetical animal study, at a dose of 0.01 mg/kg-day, the San Francisco data would suggest a risk of one in ten thousand whereas the New York data would predict that the excess risk was only two in one million.[76] Frequently, such a difference in the potential cancer risk represents the difference between whether a chemical is banned or its use encouraged. Interestingly, the UCL's on the added risk for both studies are about the same, that is 3/10,000, and this is almost 100 fold greater than that suggested by the MLE of the New York data. The point is that scientists should not be constrained by the insensitivities of the UCL methodology nor the responsiveness of the MLE; rather decisions should be heavily influenced, if not dictated, by biologic factors and good scientific judgment. Clearly, both lawyers and risk managers must be aware of the potential for a mathematical model to inadvertently over-state or underestimate the significance of the data which, at times, may have a dramatic effect on the regulatory decision.

75. An example of how low dose extrapolation models may over-respond:

RESULTS OF TESTING

	Response	
Dose (mg/kg-day)	San Francisco	New York
0	0/50	0/50
3	2/50	1/50
10	10/50	10/50

76. MLE RISK ESTIMATES

	Risk	
Dose (mg/kg-day)	San Francisco	New York
3	4/100	2/100
1	1/100	1/20,000
0.1	1/1,000	1/200,000
0.01	1/10,000	1/2,000,000

C. *Exposure Assessment*

Over the past five years, a good deal of emphasis has been placed on improving the first two steps of the risk assessment process, hazard identification and dose-response assessment. However, most health risk assessments of waste sites and other hazards which precipitate personal injury litigation are plagued by serious problems in the exposure assessment phase of the analyses. Indeed, this is the most easily mishandled of the four portions of the assessment. This is a tragedy because exposure assessment is the portion likely to be understood by the jury, the government, and the judges.

Although there have been numerous claims that exposure assessment is exceedingly difficult and uncertain, this portion contains no greater uncertainty than other steps in the process. As discussed previously, it is possible for different dose-response models to predict risks which span one to four orders of magnitude: a significant range of uncertainty. Admittedly, there are a large number of factors to consider when estimating exposure, and it is a complicated procedure to estimate the transport and distribution of a chemical which has been released into the environment. Nonetheless, the available data indicate that scientists can do an adequate job of estimating the concentration of chemicals in the environment and the resulting uptake by exposed persons if they account for the many factors that must be considered.[77]

There are at least four major pitfalls in the exposure assessment process to which one should be sensitive. First, the typical or average person, rather than the theoretical maximum exposed individual (MEI), should be the focus of a health risk assessment. Although the risk for those potentially exposed to particularly high levels needs to be understood, too much emphasis has been

77. EPA, ESTIMATING EXPOSURES TO 2,3,7,8-TCDD, 205 (1988) (Draft); Eschenroeder, Jaeger, Ospital & Doyle, *Health Risk Analysis of Human Exposures to Soil Amended With Sewage Sludge Contaminated With Polychlorinated Dibenzodioxins and Dibenzofurans*, 28 VET. HUM. TOXICOL. 435 (Oct. 1986); Paustenbach, *Important Recent Advances in the Practice of Health Risk Assessment: Implications for the 1990's*, REG. TOXICOL. PHARM. (in press) (1989); LEUNG & PAUSTENBACH, *Assessing Health Risks in the Workplace: A Case Study of 2,3,7,8-Tetrachlorodibenzo-p-dioxin*, in THE RISK ASSESSMENT OF ENVIRONMENTAL HAZARDS: A TEXTBOOK OF CASE STUDIES 689, 691 (D.J. Paustenbach ed. 1989); Bogen & Spear, *Integrating Uncertainty and Interindividual Variation in Environmental Risk Assessment*, 7 RISK ANALYSIS 427 (1987).

placed on the MEI.[78] Instead, the typical person should be the primary emphasis of the analyses even though the risk to others should also be understood. The distinction is important. If, for example, a regulatory agency bases its decision on the results of an assessment assuming that a person eats about 100 grams of fish every day of his or her lifetime (99th percentile), yet the average American eats only eighteen grams of fish per day (lifetime average), the analysis should reflect the fact that ninety-nine of 100 persons are not represented by the corresponding risk estimate.[79] To help minimize the potential for misunderstanding, it is recommended that the number of exposed persons at each of the anticipated dose levels be presented, along with the most likely and upper estimates of exposure. This has been done in only a limited number of assessments. Using an exhibit like Table 1, the risk manager or the court can readily understand the severity of the risk for each segment of the population. Provided with this information, it can then be decided whether large or small sums of money need to be expended to reduce the health risks.

The next pitfall is a variation of the first one. It involves the repeated use of conservative assumptions.[80] Several published papers have discussed this issue and have demonstrated its importance.[81] The problem can be illustrated in a recent attempt to assess the dioxin hazard posed by municipal waste incinerators. An agency evaluated the theoretical cancer risk for a child who lived within a short distance (0.8 km) from the hypothetical incinerator.[82] At first review, the analysis seemed reasonable until one noted that the child ate about two teaspoons of dirt each day, that his house was down-wind of the stack, that he ate fish from a pond near the incinerator, his fish consumption was at the ninety-fifth percentile level, he drank contaminated water from the pond, he

78. The Environmental Protection Agency has proposed guidelines on exposure related measurements for risk assessments. 53 Fed. Reg. 48,830 (Dec. 2, 1988).

79. TOLLEFSON, *Methylmercury in Fish: Assessment of Risk for U.S. Consumers*, in THE RISK ASSESSMENT OF ENVIRONMENTAL HAZARDS: A TEXTBOOK OF CASE STUDIES 845, 863 (D.J. Paustenbach ed. 1989).

80. Paustenbach, Shu & Murray, *supra* note 13, at 303; MAXIM, *Problems Associated with the Use of Conservative Assumptions in Exposure and Risk Analysis*, in THE RISK ASSESSMENT OF ENVIRONMENTAL AND HUMAN HEALTH HAZARDS: A TEXTBOOK OF CASE STUDIES 526 (D.J. Paustenbach ed. 1989); Finkel & Evans, *Evaluating the Benefits of Uncertainty Reduction in Environmental Health Risk Management*, 37 J. AIR POLL. CONTROL A. 1164 (1987).

81. Paustenbach, Shu & Murray, *supra* note 13, at 303; Finkel & Evans, *supra* note 80; Maxim & Harrington, *supra* note 13.

82. EPA, *supra* note 77.

TABLE 1

Exposure to benzene soluble organics (micrograms per cubic meter of air)[b]	People in exposure group (thousands)	Lifetime probability of lung cancer [c]	Increased in lung cancer due to coke oven emissions[a]	Number of lung cancer deaths per year due to coke oven emissions
4.5	13,900	0.0335	6.37 x 10-4	125.0
5.5	1,034	0.0344	1.49 x 10-3	22.0
6.5	54	0.0362	2.33 x 10-3	1.8
7.5	8	0.0360	3.18 x 10-3	0.4
8.9	2	0.0369	4.02 x 10-3	0.1
10.9	2	0.0389	6.04 x 10-3	0.2

a. Estimated using the Weibull probability model.
b. Background level assumed to be 3.75 micrograms per cubic meter of air.
c. Lifetime probability 0.0329 at background exposure level.

TABLE 1: The following represents one method for presenting exposure, risk, and population data. Such an approach gives risk managers all the important information needed to make the difficult decisions about where to best allocate limited resources, rather than rely on data for the maximally exposed individual (MEI). Adapted from EPA, CARCINOGEN ASSESSMENT GROUP, PRELIMINARY REPORT ON POPULATION RISK TO AMBIENT COKE OVEN EXPOSURES, 14 (1978).

ate food grown primarily from the family garden, and he drank milk from a cow which grazed on forage at the farm. This is not quite the description of a typical person living near a municipal incinerator. Regrettably, the associated upper estimate of the risk was the only one reported in the press. Certainly, it would have been more appropriate to have studied and presented the number of persons likely to be exposed to this level, as well as the level of exposure for the typical person living within ten miles of the facility. It may also have been useful to note that few farms are located near incinerators due to the need to service large communities. Without such a presentation of the data, risk managers and the public can easily be misled and, as a result, make poor decisions.

The third pitfall is to conduct an exposure assessment without considering the environmental fate of the chemical. In general, many factors such as degradation by sunlight, soil and water microbes, and evaporation will influence the degree of human exposure. For instance, the public health hazard posed by the

potential contamination of groundwater by ethanol (alcohol) washed down the sinks of taverns and restaurants was recently evaluated. It was alleged that the disposal of this listed carcinogen might place the restaurant in violation of one of California's new laws, Proposition 65. Consequently, a risk assessment was conducted. It was soon recognized that the environmental half-life of the chemical was a critical factor in this analysis. Specifically, chemicals such as methanol, ethanol, and phenol have relatively short half-lives in most waters; only about four to eight hours. This means that soon after release the ethanol would be degraded and rendered harmless by water-borne microbes or lost through volatilization, and that virtually none of the alcohol would reach the tap water of homeowners. What had been portrayed as a potentially serious hazard was shown to be insignificant when half-life was considered.

Another pitfall is to neglect to consider using biological monitoring to validate or confirm the degree of human exposure. Over the past five years, analytical chemists have increased their ability to detect very small quantities of non-natural chemicals in blood, urine, hair, feces, breath, and fat. For many chemicals, the results would be a direct indicator of either recent or lifetime exposure to a chemical. For example, the exposure to dioxin in 2,4,5-T (Agent Orange) of veterans who served in Vietnam was recently evaluated by analyzing the amount of dioxin in their blood. This study, conducted almost fifteen to twenty years after the last day of service in Vietnam, allowed epidemiologists to conclude that the vast majority of veterans had only a modest degree of exposure to dioxin; a contaminant which has been alleged to produce numerous adverse health effects in field soldiers.[83]

The last trap is the failure to validate some of the assumptions used in the analysis or the reasonableness of the results. In an attempt to position themselves so as to be above the accusation that their assessments are not sufficiently health protective, many scientists have gone overboard in selecting certain parameters used in the calculations. One example of the problem of making assumptions without checking the reasonableness, occurred during an evaluation of the cancer hazard posed by dioxin-contaminated soot from an office building fire. The risk assessment

83. Centers for Disease Control Veterans Health Studies, *Serum 2,3,7,8-Tetrachlorodibenzo-p-dioxin Levels in US Army Vietnam-Era Veterans*, 260 J. AM. MED. A. 1249 (1988).

assumed that the office workers might be exposed to the dioxin in the soot for the entire forty years that they might work in the building and that the dioxin would be released through volatilization at a particular rate. It was calculated that persons who worked forty years in the office building would be exposed to an increased cancer risk much greater than 1 in 1,000,000, and as a result, the building was not reoccupied. Even if one agreed with the assertion that an increased cancer risk of 1 in 1,000,000 is the maximum risk to which one should be exposed, something in the analysis seemed flawed. After some study, it was shown that the assumption regarding dioxin's volatility was too conservative. Apparently, no one checked to see if the volatilization rate was reasonable. Specifically, had this assumption been accurate, the dioxin would have all been volatilized and been removed via the ventilation system only four years after reoccupation. In short, the exposure assessment assumed exposure was to occur for forty-six years even though it would not have been present after four years. The moral is that in any assessment a validation should be performed to insure that the assumptions and results are reasonable.[84]

D. *Risk Characterization*

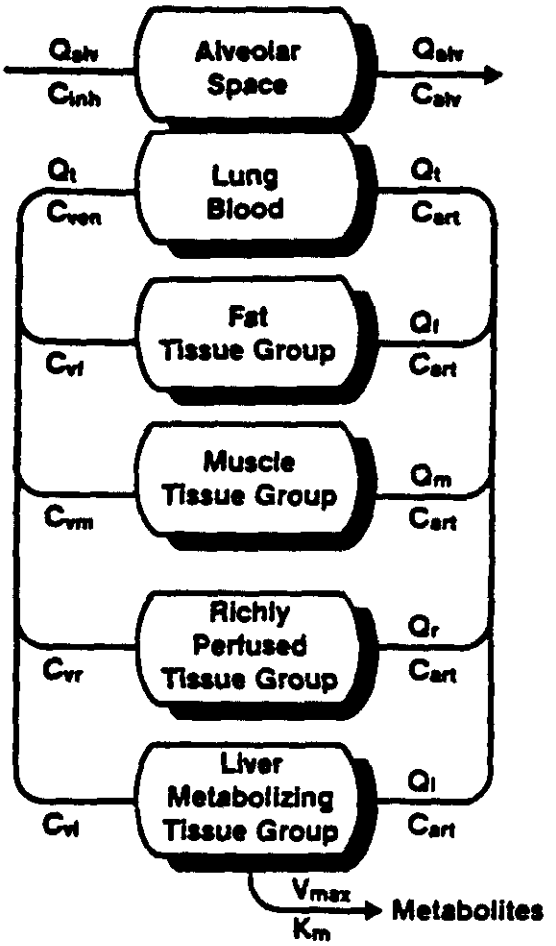
The final step in a risk assessment, risk characterization, also contains potential pitfalls.[85] Among the most frequent shortcomings is to portray the theoretical increased cancer risk of one in a million as a serious public health risk. First, it is important to remember that these usually represent the upper estimate of the potential risks, not a true estimate of risk. Indeed, as stated in nearly every risk assessment conducted by a regulatory agency, "These estimates represent an upper bound of the plausible risk and are not likely to underestimate the risk. The actual risk may be much lower, and in some cases, zero."[86] In short, unless the estimate is based on the results of a PB-PK scale-up procedure and a biologically-based model that tries to incorporate all the pertinent biologic data, the risk estimates are more applicable to a rat than a human (Figure 5).

84. Maxim, *supra* note 80.

85. Slovic, *Perception of Risk*, 236 SCIENCE 280 (1987); Wilson & Crouch, *Risk Assessment and Comparisons: An Introduction*, 236 SCIENCE 267 (1987).

86. *See, e.g.*, EPA, HEALTH ASSESSMENT DOCUMENT FOR TRICHLOROETHYLENE A-120 (1982), EPA, HEALTH ASSESSMENT DOCUMENT FOR DICHLOROMETHANE 5-94 (draft) (1983).

FIGURE 5



Where:

Q_{alv}	Alveolar ventilation rate (liters air/hr)
C_{inh}	Concentration in inhaled air (mg/liter air)
C_{alv}	Concentration in alveolar air (mg/liter air)
C_{exh}	Concentration in exhaled air (mg/liter air)
N	Blood air partition coefficient (liters air/liter blood)
Q_t	Cardiac output (liters blood/hr)
C_{art}	Concentration in arterial blood (mg/liter blood)
C_{ven}	Concentration in mixed venous blood (mg/liter blood)
V_{max}	Maximum enzymatic reaction rate (mg/hr)
K_m	Michaelis constant for enzymatic reaction (mg/liter blood)
Q_i	Blood flow rate to tissue group (liters blood/hr)
V_i	Volume of tissue group (liters i)
C_i	Concentration in tissue group (mg/liter i)
A_i	Amount in tissue group (mg)
C_{vi}	Concentration in venous blood leaving tissue group (mg/liter blood)
P_i	Tissue: blood partition coefficient (liters blood/liter i)

FIGURE 5: A physiologically-based pharmacokinetic (PB-PK) model as developed by Ramsey & Anderson, *A Physiologically-Based Description of the Inhalation Pharmacokinetics of Styrene in Rats and Humans*, 73 TOXIC'L. AND APP. PHARM. 159, at 160 (1984). These types of models allow scientists to predict how humans will respond to a chemical based on data collected in rodents. Basically, the movement and transformation of the test chemical within the rodent is described by mathematical equations. The same is done for the human. By comparing the two, one can quantitatively predict the human response. This methodology has only been used by toxicologists [illegible]

Central to the area of risk characterization is the accurate and unbiased presentation of the significance of the data. Specifically, regulatory agencies have been subject to the pitfall of stating that the results of low-dose models can actually predict the increased cancer risk for exposed individuals. As recently discussed by Dr. Frank Young,[87] the current Commissioner of the FDA, this was not the intent of such estimates:

> In applying the de minimis concept and in setting other safety standards, FDA has been guided by the figure of "one in a million." Other Federal agencies have also used a one in a million level such as the Occupational Safety and Health Administration and the Environmental Protection Agency. Both agencies rely on the one in one million increased risk over a lifetime as a reasonable criterion for separating high-risk problems warranting agency attention from negligible risk problems that do not. The risk level of one in one million is often misunderstood by the public and the media. It is not an actual risk - i.e., we do not expect one out of every million people to get cancer if they drink decaffeinated coffee. Rather, it is a mathematical risk based on scientific assumptions used in risk assessment. FDA uses a conservative estimate of risk to ensure that the risk is not understated. We interpret animal test results conservatively and we are extremely careful when we extrapolate risks to humans. When FDA uses the risk level of one in one million, it is confident that the risk to humans is virtually nonexistent.

Frequently, regulators suggest that most environmental regulations have been promulgated so as to keep the theoretical cancer risks below one in a million. In fact, the theoretical risks associated with currently enforced environmental regulations are in the vicinity of one in 100,000, not one in 1,000,000.[88] Occupational exposure limits usually have theoretical risks in the region of one in 1,000.[89]

We should also attempt to present the significance of these risks in a more understandable fashion. For example, the goal of some environmental standards, such as the maximum contaminant levels (MCL) for drinking water, is to keep the maximum plausible risk to about one in 1,000,000. What few persons rec-

87. Young, *Risk Assessment: The Convergence of Science and the Law*, 7 REG. TOXICOL. PHARM. 179, 184 (1987).

88. Travis, Richter, Crouch, Wilson & Klema, *supra* note 18, at 416-18 (a table of risk levels for 132 chemicals regulated by government agencies); Travis & Hattemer-Frey, *supra* note 20, at 875 (a table of upper-bound risk levels after regulation of 36 chemical carcinogens).

89. Rodricks, Brett & Wrenn, *supra* note 20, at 315.

ognize is that since the incidence of cancer in the population is currently about 25%, this is equivalent to insuring that the lifetime cancer risk for any person exposed to this level of contamination is not greater than 250,001 in 1,000,000 (25.0001%) rather than 250,000 in 1,000,000. If society demands this standard of care, that is its choice. However, both society and its risk managers deserve to understand the significance of the risk before deciding to spend money on one hazard versus another.

Many news releases of the past ten years seem to indicate that agencies have demanded that exposure to chemicals must be controlled to a level that risks are only in the vicinity of one in 1,000,000. However, recent work has shown that this has clearly not been the case.[90] Specifically, we have tended to allow exposure levels to be influenced by the number of exposed individuals (Figure 6).

V. Conclusion

What does all of this mean to the legal profession? If one considers all of the issues raised here, the reasons why risk assessments are important and necessary becomes clear. The process gives non-scientists the insight and knowledge needed to make more objective and rational decisions in a complex scientific arena. Assessments give regulators and courts the information needed to know whether a particular hazard poses a significant or de minimis risk.[91] The hope is that this insight will result in cost effective decisions and fair court settlements.

In toxic tort cases, risk assessments can clearly play an important role. Risk assessments can help substantiate medical opinions regarding causation, quantitatively describe the likely degree of exposure, reduce reliance on experts' professional intuitions, and neutralize subjective or unsubstantiated claims about exposure level and associated health risks. My experience is that attorneys who have been aware of the benefits of the risk assessment process have done very well in representing their clients in cleanup and personal injury litigation.[92] This has, in part, been be-

90. *See, e.g.*, Travis, Richter, Crouch, Wilson & Klema, *supra* note 18.

91. C. Whipple, *Dealing with Uncertainty About Risk in Risk Management*, in Hazards: Technology and Fairness 44, 45 (National Academy Press 1986).

92. Black, *Evolving Legal Standards for the Admissibility of Scientific Evidence*, 239 Science 1508 (1988); Mitchell, Ward & Grutsch, *Legal Standards of Causation in Chemical Exposure Litigation*, 7 Reg. Toxicol. Pharm. 206, 211 (1987).

FIGURE 6
EFFECT OF INDIVIDUAL VS. POPULATION RISK ON CHEMICAL CARCINOGEN REGULATION

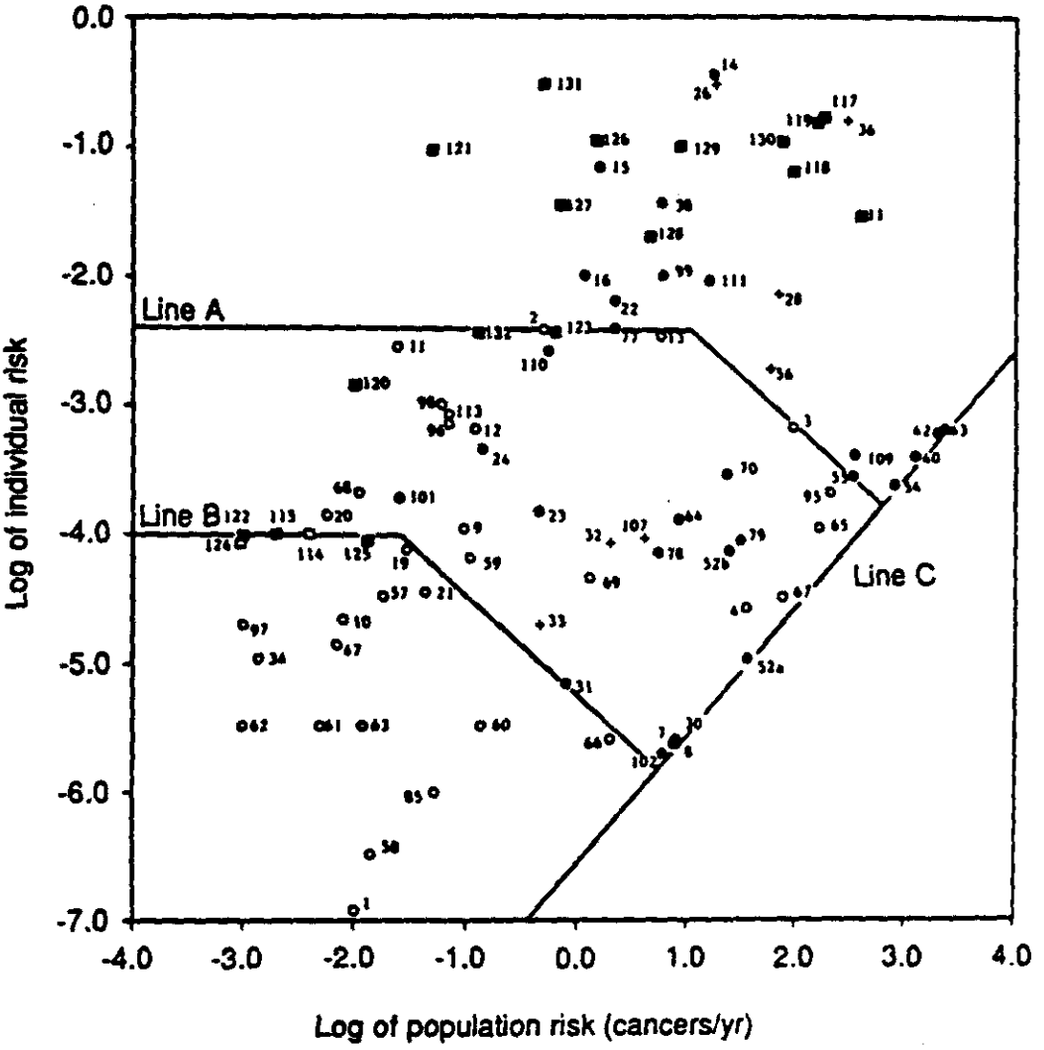


FIGURE 6. A compilation of the individual risk levels inherent in various regulatory decisions as a function of the number of exposed persons. Note that when the number of exposed persons is relatively small, the allowable level of exposure increases. From Travis, Richter, Crouch, Wilson, and Klema, *supra* note 18, at 419.

cause high quality assessments have helped juries quantitatively evaluate the reasonableness of the medical claims. An understanding of the pitfalls and shortcomings that have been identified and discussed here should give a significant advantage to attorneys and scientists who must respond to or present health risk assessments.

NYT 10/22/91

THE NEW YORK TIMES,

U.S. Said to Lack Data on Threat Posed by Hazardous Waste Sites

By KEITH SCHNEIDER

Special to The New York Times

WASHINGTON, Oct. 21 — The National Research Council said today that the nation's mammoth program to clean up toxic waste was hampered by its inability to tell the difference between dumps posing a real threat to human health and those that do not.

In a report, the research council, an arm of the National Academy of Sciences, said that because not enough money was spent on developing a sound scientific system for setting priorities, the nation faced the prospect of wasting billions of dollars on dumps that posed little or no risk and ignoring dumps that were a true threat to the environment and public health.

The report is the latest in a succession of studies in and out of Government to identify weaknesses in the management of the Superfund program, the Federal and industry-financed project begun in 1980 to clean up abandoned chemical waste dumps.

> 'We shouldn't be making decisions on spending billions of dollars out of ignorance.'

But its recommendations are equally applicable to even more expensive cleanup programs managed and paid for by the Department of Energy and the Department of Defense. The two departments are spending more than $6 billion this fiscal year on cleaning up toxic chemical and radioactive waste sites.

Need to Evaluate Risks

"We shouldn't be making decisions on spending billions of dollars out of ignorance," said Dr. Thomas C. Chalmers, distinguished physician of the Department of Veterans Affairs in Boston, a member of the committee that prepared the report. "We need much more data to determine which sites ought to be pursued and we need to set up a better system of evaluating risks."

By any measure, according to the research council's study, the task of sopping up the poisonous byproducts of 20th century industrialism is monumental. According to a Congressional study, more than 400,000 leaking chemical storage tanks, pesticide dumps, piles of mining wastes, underground tanks and waste pits exist around the country.

Prompted by the public's concern that such sites leaked poisons into the air and water and posed grave risks to communities, Congress established the Superfund program, the first public works program that focused on environmental restoration.

But the program has been hampered from its inception by the difficulty of the work, ineffective management and poor cost controls. Of the more than 1,200 toxic waste sites designated by the Government to be the most dangerous, only 63 have been cleaned up, the Environmental Protection Agency reports.

The study was ordered by the National Academy of Sciences, a private organization that was chartered by Congress to examine science and technology issues at the request of Federal agencies.

With its new study, the research council has identified another basic problem with the Superfund program: a striking lack of scientific data. The research council found that the Government had no comprehensive inventory of toxic waste sites, no program for finding new sites and almost no sound scientific data for determining how people are affected by their exposure to low levels of chemicals leaking into the air and water from waste sites.

The report said, "A decade after implementation of Superfund, and despite Congressional efforts to redirect the program, substantial public health concerns remain, and critical information on the distribution of exposures and health effects associated with hazardous waste sites is still lacking."

Almost nothing is known about the effects on human health of most chemicals found in hazardous waste sites, the study said. Most people exposed to hazardous waste at those sites come in contact with minute amounts of chemicals, but very little is known about how they are affected, it continued. Another gap in the Government's data is that scientists have virtually no idea of the risks posed by two or more chemicals that react in a waste site to form another toxic compound.

In the absence of scientific and health data, the Environmental Protection Agency has developed separate methods for making estimates about the risk of a toxic waste site that help guide the agency's priorities for cleaning them up.

Sites that the agency knows contains large amounts of liquid chemicals, are close to population centers and are leaking into underground sources of water generally receive the most attention. The agency also considers sites nearest to rivers and flood plains to be the most dangerous to communities.

"We are spending hundred of million of dollars here and throughout the Government to get more data to improve health studies, to understand how chemicals move in the environment, and find out what the exposures are," said Richard J. Guimond, the national Superfund director at the E.P.A. "But there are 40 million people out there who live within four miles of a Superfund site. Do you tell them, 'Hey, sit tight. We're studying this problem. Until we have better information in 20 years we won't take action?' "

The authors of the report, an eight-member committee of experts from Federal and state health agencies and prominent universities, called on Congress and the Bush Administration to sharply increase the amount of money being spent on research to develop new technical tools that would quickly and accurately identify the risks from exposure to chemicals in toxic waste dumps. Less than 1 percent of the Federal money spent on toxic waste cleanups is directed toward such research, the study said.

Recommended Dietary Allowances

10th Edition

Subcommittee on the Tenth Edition of the RDAs
Food and Nutrition Board
Commission on Life Sciences
National Research Council

NATIONAL ACADEMY PRESS
Washington, D.C. 1989

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Richard J. Havel, *Chairman*
Food and Nutrition Board and
the Subcommittee on the Tenth
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Recommended Dietary Allowances

10th Edition

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COPPER

Copper is an essential nutrient for all vertebrates and some lower animal species (Davis and Mertz, 1987). Several abnormalities have been observed in copper-deficient animals, including anemia, skeletal defects, demyelination and degeneration of the nervous system, defects in pigmentation and structure of hair or wool, reproductive failure, myocardial degeneration, and decreased arterial elasticity. There are a number of important copper-containing proteins and enzymes, some of which are essential for the proper utilization of iron (Davis and Mertz, 1987).

Assessment of Copper Status

Although hypocupremia is readily produced in animals during experimental copper deficiency, circulating copper concentration is not necessarily a valid index of copper nutriture in humans (Solomons, 1979). Ceruloplasmin, a protein-copper complex, is strongly influenced by hormonal changes or inflammation, thus limiting its usefulness as an indicator (Mason, 1979). Determination of erythrocyte superoxide dismutase (SOD) activity appears to be a promising technique for assessing copper status in humans (Uauy et al., 1985).

Evidence for Human Requirement

Severe copper deficiency is rare in human beings (Cartwright and Wintrobe, 1964; Danks, 1988). Copper depletion sufficient to cause hypocupremia has been observed during total parenteral nutrition (Shike, 1984) and in cases of Menkes' steely hair disease—a rare, inherited disease resulting in impaired copper utilization (Menkes et al., 1962). The hypocupremia reported in protein-calorie malnutrition, sprue, nephrotic syndrome, and certain other diseases is probably unrelated to dietary copper intake and is believed to be secondary to a state of hypoproteinemia and inability to provide adequate amounts of the aproprotein for ceruloplasmin synthesis (Mason, 1979). Under normal circumstances, dietary copper deficiency is not known to occur in adults, but it has been observed in malnourished children in Peru; its manifestations are anemia, neutropenia, and severe bone demineralization (Cordano et al., 1964). In the early 1970s in the United States, similar findings were recognized in a few very small premature infants who were hospitalized for long periods and exclusively fed modified cow's milk formula or received prolonged parenteral alimentation. Presumably, these aberrations reflected a deficient dietary intake of copper (Cordano, 1974). More recently, copper deficiency has been shown to impair the growth of Chilean infants recovering from malnutrition (Castillo-Duran and Uauy, 1988).

The concentration of copper in the human fetus increases substantially during gestation, about half of the total fetal copper accumulating in the liver (Widdowson et al., 1974). These hepatic reserves are believed to protect the full-term infant against copper deficiency during the first few months of life. In the United States, tissue copper concentrations remain remarkably steady throughout adult life (Schroeder et al., 1966). The relatively constant copper concentrations in most tissues indicate sufficient dietary intake and effective homeostatic control of copper.

Epidemiological and experimental animal studies suggest a positive correlation between the zinc-to-copper ratio in the diet and the incidence of cardiovascular disease (Klevay, 1984). Elevated plasma cholesterol levels, impaired glucose tolerance, and heart-related abnormalities have been observed in some human subjects consuming only 0.8 to 1.0 mg copper per day (Klevay et al., 1984; Reiser et al., 1985), but not in others (Turnlund et al., 1989).

Dietary Sources and Usual Intakes

Organ meats, especially liver, are the richest sources of copper in the diet, followed by seafoods, nuts, and seeds. The concentration of copper in drinking water is highly variable; it is much influenced by the interaction of the water's acidity with the piping system. Additional contributions to intake may come from adventitious sources, such as copper-containing fungicides sprayed on agricultural products. Human milk contains approximately 0.3 mg/liter; cow's milk only about 0.09 mg/liter (Varo et al., 1980)

Older analytical data indicating that most U.S. diets provide a daily copper intake between 2 and 5 mg are now being reexamined and questioned (Klevay, 1984). The Total Diet Study, based on the extensive dietary analyses performed by the U.S. Food and Drug Administration, showed that the daily intake of copper for adult males and females averaged about 1.2 and 0.9 mg, respectively, from 1982 to 1986 (Pennington et al., 1989). The intakes for infants 6 to 11 months old and toddlers 2 years old were 0.45 and 0.57 mg daily.

Bioavailability

Several different factors may affect the bioavailability of dietary copper. Jacob et al. (1987) observed that high intakes of vitamin C (605 mg/day) decreased serum ceruloplasmin but had no effect on overall body copper status. Zinc intakes slightly above RDA levels reduced apparent copper retention in young men and adolescent females (Festa et al., 1985; Greger et al., 1978). The degree of copper deficiency may be influenced by the type of carbohydrate consumed, since rats fed a diet containing fructose developed more severe signs of copper deficiency than did rats fed a diet containing either glucose or starch (Fields et al., 1984). Although it may be assumed that the interaction between copper and ascorbic acid involves reduction and chelation of the metal in the intestine, the nature of the interaction of copper with zinc or carbohydrates is not yet known.

Estimated Safe and Adequate Daily Dietary Intakes

Adults In the past, estimates of the copper requirement for humans were derived from metabolic balance studies. However, the balance technique can lead to false estimates of nutritional requirements because the efficiency of copper absorption is increased or decreased in response to low or high copper intakes, respectively (Turnlund et al., 1989). Older balance studies suggested that the adult requirement for copper ranged from 2.0 to 2.6 mg/day, whereas later studies indicated that intakes less than 2.0 mg/day, and often not much more than 1.0 mg/day, could maintain positive copper balance (Mason, 1979). In a recent metabolic ward study, 13 men consuming a variety of typical U.S. diets were found to need 1.30 mg/day to replace fecal and urinary losses (Klevay et al., 1980).

Whole-body surface losses of copper are highly variable. Such variability makes it difficult to select an appropriate value for the losses incurred through this pathway, but recent estimates indicate that copper losses from the body's surface are less than 0.1 mg/day (Turnlund et al., 1989). If the true gastrointestinal absorption of copper at intakes of 1.7 to 2.0 mg is 36% (± 1.3 SEM) (Turnlund et al., 1989), then a dietary intake of 0.3 mg/day is required to replace body surface losses. Adding this figure to the average dietary intake of 1.3 mg/day needed to replace urinary and fecal losses indicates that a total dietary copper intake of approximately 1.6 mg/day is required to maintain balance in adult men.

Many U.S. diets provide less than 1.6 mg of copper daily (Klevay, 1984). Since anemia or neutropenia ascribable to copper deficiency has not been observed in adults consuming typical U.S. diets, there is an obvious discrepancy between the experimentally derived copper requirement as defined by balance studies and currently estimated dietary copper intakes. This suggests either a long-term homeostatic adaptation to low copper intakes, or an incorrect estimate of dietary copper intake due to the underreporting of certain foods and water that are sources of the element. Because of the uncertainty about the quantitative human requirement for copper, it is not possible to establish an RDA for this trace element. Rather, the subcommittee recommends 1.5 to 3 mg/day as a safe and adequate range of dietary copper intake for adults.

Infants and Children The average daily intake of copper by exclusively breastfed North American infants was 0.23 ± .07 mg over the first 4 months of lactation (Butte et al., 1987), or approximately 40 ± 16 μg/kg per day. This intake is substantially less than the 80 μg/kg per day recommended by a World Health Organization Expert Committee (WHO, 1973), but approaches the lower limit of the estimated requirement range of 45 to 135 μg/kg per day suggested by Cordano (1974) for rapidly growing infants with poor stores. Positive copper balance has been observed in normal children ages 3 months to 8 years with intakes as low as 35 ± 22 μg/kg per day (Alexander et al., 1974).

Studies in animals have shown high bioavailability of copper from human milk (Lönnerdal et al., 1985). Furthermore, the sizeable hepatic copper reserve built up during fetal development appears to contribute to the early needs of the growing full-term infant (Widdowson et al., 1974). After 3 months of age, the recommended copper intake of 75 μg/kg/day translates into dietary ranges of 0.4 to 0.6 and 0.6 to 0.7 mg/day for reference infants from birth to 6 months and from 6 to 12 months old, respectively. The introduction of solid foods at 4 to 6 months of age should enable the older infant fed a mixed diet to meet the copper recommendations (Gibson and De Wolfe, 1980), but the exclusively breastfed infant will have difficulty in achieving those levels because copper levels in human milk decline from 0.6 to 0.2 mg/liter during the first 6 months of lactation (Vuori and Kuitunen, 1979). These recommended intakes may be inadequate for the premature infant, who is always born with low copper stores (Shaw, 1973).

The American Academy of Pediatrics has recently recommended that infant formulas provide 60 μg of copper per 100 kcal (AAP, 1985). By following this recommendation, a typical formula-fed in-

fant from birth to 6 months of age receiving 700 kcal per day would consume approximately 0.4 mg of copper per day.

In preadolescent and adolescent girls, fecal and urinary losses were at or near equilibrium with a dietary copper intake of 1 to 1.3 mg/day (35 to 45 µg/kg body weight per day) (Engel et al., 1967; Greger et al., 1978; Price and Bunce, 1972). The recommended copper range of 1.0 to 2.0 mg/day for 7- to 10-year-old children provides at least 40 µg/kg body weight/day.

Excessive Intakes and Toxicity

An FAO/WHO Expert Committee concluded that no deleterious effects can be expected in humans whose copper intake is 0.5 mg/kg body weight per day (FAO/WHO, 1971). Usual diets in the United States rarely supply more than 5 mg/day, and an occasional intake of up to 10 mg/day is probably safe for human adults. Although storing or processing acidic foods or fluids in copper vessels can add to the daily intake, overt toxicity from dietary sources is extremely rare in the U.S. population (NRC, 1977)

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MANGANESE

Manganese has been shown to be an essential element in every animal species studied. Signs of deficiency include poor reproductive performance, growth retardation, congenital malformations in the offspring, abnormal formation of bone and cartilage, and impaired glucose tolerance (Hurley and Keen, 1987). Several enzymes, such as decarboxylases, hydrolases, kinases, and transferases, are nonspecifically activated by manganese *in vitro*. There are two known manganese metalloenzymes: pyruvate carboxylase and superoxide dismutase, both localized in mitochondria.

Manganese deficiency has never been observed in noninstitutionalized human populations because of the abundant supply of manganese in edible plant materials compared to the relatively low requirements of mammals (Underwood, 1981). Analyses of a variety of tissues taken from humans of various ages have indicated that there is no tendency for either a decrease or an increase in manganese accumulation throughout most of the life cycle (Schroeder et al., 1966). This constancy of manganese concentration in the tissues suggests adequate dietary intake coupled with strong homeostatic control. There has been only one recorded case of a possible manganese deficiency in a human—a male subject in a vitamin K deficiency study who was fed a purified diet from which manganese was inadvertently omitted (Doisy, 1973). His total diet (food and water) furnished only about 0.35 mg of manganese per day. Retrospective analyses revealed that there were 55 and 95% declines in his serum and stool manganese levels, respectively, over a 17-week period (Doisy, 1974).

Progress in the field of manganese nutrition has been hampered because of the lack of a practical method for assessing manganese status. Blood manganese levels appear to reflect body manganese status of rats fed deficient or adequate amounts of manganese (Keen et al., 1983), but consistent changes in blood or plasma manganese levels have not been observed in depleted or repleted human subjects (Freeland-Graves et al., 1988; Friedman et al., 1987). Animal studies have shown that the activity of mitochondrial superoxide dismutase is a function of dietary manganese intake, but practical usefulness of this enzyme as an indicator is uncertain since tissues containing mitochondria are generally not readily available for nutritional status assessment purposes.

Dietary Sources and Usual Intakes

Whole grains and cereal products are the richest dietary sources of manganese, and fruits and vegetables are somewhat less so. Dairy products, meat, fish, and poultry are poor sources. Tea is a rich source of manganese, but typical drinking water consumed at the rate of 2 liters daily contributes only about 40 to 64 μg, or about 2 to 3% of the amount furnished by diet (NRC, 1980).

Although there is now a body of data concerning the levels of manganese in the diet, little is known about the chemical form or nutritional bioavailability of the manganese in foods (Kies, 1987). Extreme dietary habits can result in manganese intake outside the provisionally recommended limits; consumption of a varied and balanced diet will reliably furnish safe and adequate amounts.

The Total Diet Study conducted in the United States between 1982 and 1986 indicated that the mean daily dietary manganese intake was 2.7 and 2.2 mg for adult men and women, respectively (Pennington et al., 1989). Teenage boys consumed an average of 2.8 mg/day, whereas girls consumed only 1.8 mg/day. Mean manganese intakes were 1.1 and 1.5 mg/day for 6- to 11-month-old babies and 2-year-old toddlers, respectively.

Estimated Safe and Adequate Daily Dietary Intakes

Adults Several short-term balance studies in adult humans fed different amounts of manganese have been conducted in an attempt to define the requirement for this trace element (reviewed by Freeland-Graves et al., 1987). However, there are many problems with using the balance method to estimate trace element requirements (Freeland-Graves et al., 1988). At best, such studies determine the

Protecting the Environment

Murray Weidenbaum

Every poll of citizen sentiment shows overwhelming support for doing more to clean up the environment. A public opinion survey by *The New York Times* and CBS News reported in 1983 that 58 percent of the sample agreed with the following statement: "Protecting the environment is so important that requirements and standards cannot be too high and continuing environmental improvements must be made regardless of cost."

Despite the continuation of such an overwhelming public mandate and a plethora of new laws and directives by the EPA (Environmental Protection Agency) plus hundreds of billions of dollars of compliance costs expended by private industry, the public remains unhappy with the results.

Unfortunately, environmental action is an extremely important example of not wishing to pay the piper. Those same citizens who want environmental improvements "regardless of cost" vociferously and adamantly oppose the location of any hazardous-waste facility in their own neighborhood. Nor are they keen on paying for the cleanup. Of course, they strongly favor cleaning up the environment, but each prefers to have the dump site located in someone else's backyard and to have the other fellow pay for it.

An example of this situation is the reaction of the enlightened citizens of Minnesota to a $3.7 million grant from the EPA to build and operate a state-of-the-art chemical landfill that could handle hazardous wastes with a high assurance of safety. In each of the 16 locations that the state proposed, the local residents raised such a fuss and howl that the state government backed off. Ultimately, the unspent grant was returned to the EPA.

The Minnesota experience is not exceptional. The EPA was also forced to stop a project to test whether the sludge from a municipal waste treatment plant could be used as a low-cost fertilizer. Public opposition was fierce, even though the EPA was going to use federally owned land and the sludge was expected to increase crop yields by 30 percent.

Since 1980, not a single major new disposal facility has been sited anywhere in the United States. According to a state-by-state review, the outlook for the future is "even more bleak," in large part because of the deteriorating emotional atmosphere surrounding any effort to locate a new dump site. As Peter Sandman of Rutgers University has pointed out, the public perceives environmental matters not only emotionally, but also morally. "Our society," he has written, "has reached near-consensus that pollution is morally wrong – not just harmful or dangerous ... but wrong." Yet, the individuals who make up that same public are reluctant to personally assume the burdens associated with that strongly held view.

This ambivalent attitude toward the environment is not new. In 1969, the National Wildlife Federation commissioned a national survey to determine how much people were willing to pay for a cleaner environment. At a time of peak enthusiasm for environmental regulation, the public was asked, "To stop pollution destroying our plant life and wildlife, would you be willing to pay an increase in your monthly electric bill of $1?" The "no" vote won hands down, 62 percent to 28 percent (with 10 percent "not sure"). That study, we should recall, was taken before the big runup in utility bills. Perhaps not too surprisingly, the survey showed strong support for taxing business to finance environmental cleanup.

In other words, most Americans very much want a cleaner environment, but are neither willing to pay for it nor to inconvenience themselves. Americans try to take the easy way out – by imposing the burden

on "someone else," preferably a large, impersonal institution.

It is much easier for Congress to express a desire for cleaner air or purer water than for an agency like the EPA to fulfill that desire. Vast sums of money have been spent for these purposes in recent years. From 1970 to 1986, Congress appropriated more than $55 billion for the operation of the EPA. The head-count of EPA employment rose from a few hundred in 1970 to over nine thousand in 1988. These numbers are dwarfed by the costs for the private sector to comply with government's rules on environmental cleanup. The U.S. Council on Environmental Quality estimated the total at more than $100 billion for 1988, and more than $750 billion for the preceding decade (in dollars of 1986 purchasing power).

These staggering outlays have not prevented critics from initiating an almost endless array of lawsuits whose main purpose is to get the EPA to act faster and to do more. Typical of the assaults on the EPA is this statement by Congressman James J. Florio of New Jersey: "They are not in charge. They do not have the resources by their own actions to get the work done, and they are more interested in cosmetics than anything."

The plaintive response of the EPA administrator at the time was that "EPA's plate is very full right now." That plate is being heaped higher on an almost daily basis. One of EPA's newest responsibilities, for instance, is regulation of genetically engineered pesticides. Rapid scientific improvements permit the detection and, perhaps, regulation of ever more minute quantities of pollutants.

Meanwhile, John and Jane Q. Public) are making the problem worse. In 1965, the average American disposed of three pounds of garbage a day. By 1985, that figure was up to four pounds each day and rising – in addition to wastes from agriculture, mining, industry, construction and demolition, sewage, and junked autos.

The EPA can claim important accomplishments. Between 1970 and 1985, air pollution from vehicles fell by 46 percent for hydrocarbons, 34 percent for carbon monoxide, and 75 percent for lead. Rivers that were nearly devoid of life teem with fish once again. Lake Erie, so laden with pollutants in 1969 that a river feeding into it caught fire, has been revived.

Despite these successes, the EPA frequently falls short in meeting congressionally mandated goals for pollution cleanup. The hard fact is that the status quo in environmental policy is not sufficient. Congress continues to pass high-sounding legislation with unrealistic timetables and inflexible deadlines, while the EPA gets ever greater responsibility and private industry spends billions more on environmental compliance. In the words of the EPA's former administrator, William Ruckelshaus, "EPA's statutory framework is less a coherent attack on a complex and integrated societal problem than it is a series of petrified postures."

The Public Sector Drags its Feet

Misperceptions of the villains in the pollution story abound. Many people fall into a common trap – that of associating polluters exclusively with business. Many companies do generate lots of pollution. But the same can be said about government agencies, hospitals, schools, and colleges.

The EPA lacks the enforcement power over the public sector that it possesses over the private sector. Reports of plant closings because of the high cost of meeting environmental standards are common. In contrast, there is no record of a single government facility closing down because it was not meeting ecological requirements.

It is not surprising that the GAO (General Accounting Office) says that the performance of federal agencies in the disposal of hazardous waste "has not been exemplary." A GAO report issued in 1986 says that, of 72 federal facilities inspected, 33 were in violation of EPA requirements and 22 had been cited for Class 1 (serious) violations. Sixteen of the thirty-three facilities remained out of compliance for six months or more. Three had been out of compliance for more than three years. A follow-up report by the GAO in 1987 showed little further progress. Only four of eleven federal agencies had completed the identification of hazardous-waste sites and none had finished assessing the environmental problems they had uncovered. Of 511 federal sites failing to meet EPA standards, only 78 had been cleaned up.

A major offender is the DOD (Department of Defense), which now generates more than 500,000 tons of hazardous waste a year. That is more than is produced by the five largest chemical companies combined. The lax situation uncovered by the GAO at Tinker Air Force Base, in Oklahoma, is typical of the way in which many federal agencies respond to the EPA's directives: "Although DOD policy calls for the military services to...implement EPA's hazardous waste management regulations, we found that Tinker has been selling...waste oil, fuels, and solvents rather than...recycling."

The GAO reported that two of the five commercial waste sites receiving the base's wastes had major compliance problems. Also, personnel at Tinker Air Force Base were dumping hazardous wastes in landfills that themselves were in violation of EPA requirements. In one case, the EPA had been urging the Oklahoma Department of Health for several years not to renew a landfill's permit. In another instance, the State Water Resources Board was seeking a court order to close the site. Civilian agencies, including those in state and local governments, continue to be reluctant to follow the same environmental standards that they impose on the private sector.

Environmental action is an important example of not wishing to pay the piper

Federal policy arbitrarily excludes one of the largest single sources of pollution from the EPA's effective jurisdiction: the runoff of pesticides and fertilizers from farms. The EPA reports that in six of the agency's ten regions, pollution from farms and urban streets is the principal cause of water quality problems. But pollution from these sources remains virtually unregulated.

Large quantities of agricultural pollution can be controlled fairly easily at low cost by using limited-till plowing techniques. In striking contrast, industrial pollution control has often been pushed to the limits of economic feasibility. Congress follows a double standard: for urban and industrial pollution it requires the imposition of tough standards to qualify for permits to discharge wastes. For rural and farm pollution, the EPA is merely given money to study the problem.

Congress wants a cleaner environment. But so far it has not mustered the will required to impose the most modest pollution controls on a politically powerful group of constituents. Farm families also want a cleaner environment – but it is always nice to get someone else to pay for your desires.

Economic Solutions to Hazardous Waste Problems

Turning to specific environmental problems, we can start with the controversy over the disposal of hazardous wastes. Instances of toxic-waste contamination at Love Canal, in New York State, and at Times Beach, Missouri, have brought a sense of urgency to the problem. The public mood on the subject of hazardous waste leaves little room for patience – but much opportunity for emotional response.

Emotionally charged responses are encouraged by the fact that even scientists know little about the effects on human health of many toxic substances such as the various forms of dioxin. The EPA can now measure levels of some substances in terms of parts per billion and occasionally per quadrillion, but even the experts still debate the significance of exposure at those rates. The scare headlines about chemical health hazards deal with exposures that are akin to the proverbial needle in the haystack. Actually, the needle-haystack comparison is much too modest. One part per billion is the equivalent of one inch in 16,000 miles, a penny in $10 million, four drops of water in an Olympic-size pool, or a second in 32 years.

The most severe reaction to dioxin reported so far by humans is a bad case of chloracne, a severe acne-like rash. The bulk of the available information on dioxin and other hazards is based on extrapolating from data on animal experiments, which is very tricky. Most tests on animals are conducted at extremely high concentrations of the suspected element, which do not reflect real-world conditions in which the animals (or humans) live. Scientists note that the massive doses that are fed the animals overwhelm their entire bodies. A level of exposure that is harmful to one type of animal may not be injurious to another. The lethal dose of the most toxic dioxin (2,3,7,8, TCDD) for hamsters is 5,000 times higher than that for guinea pigs. Extrapolating the results to humans involves even more conjecture. Still, our hearts must go out to the people in Times Beach, Missouri, and in Love Canal, New York, who have suffered severe financial and psychological damage from the emotional responses to the scare stories they have seen and heard so frequently.

In trying to avoid a repetition of these situations, the EPA has promulgated detailed regulations on how polluters must keep track of hazardous wastes and how they should dispose of them. Because of growing public concern over leaky and dangerous dump sites, Congress in late 1986 extended and expanded Superfund, the program designed to clean up hazardous-waste sites. The law requires companies and, ultimately, consumers, to pay $9 billion into Superfund by 1991. Yet, despite all this effort and attention, the problem of how to dump hazardous wastes is scarcely less serious than it was in 1980, before Congress passed the original Superfund law.

As it stands, the law provides for a large fund raised primarily through taxes on producers of chemical and petroleum products. The EPA uses this money to identify and clean up hazardous waste sites. But little progress is made because, as we noted earlier, there is a severe shortage of dump sites.

Economic Incentives Needed

A more clearheaded view of waste disposal problems is needed in the United States. Because definitions vary among levels of government, estimates of the amount of hazardous waste disposed of each year in the United States range from 30 million to 264 million metric tons. Most of this waste is buried in landfills because incineration, the safest and most effective means of disposal, is nearly ten times as costly. Even so, government and industry spend more than $5 billion each year to manage toxic wastes. The annual cost by 1990 is projected to reach $12 billion.

Rivers from coast to coast that were nearly devoid of life teem with fish once again

Many experts believe that using landfills is inherently unsafe, if for no other reason than that they are only storage sites. Moreover, there are not enough of them. The EPA estimates that 22,000 waste sites now exist in the United States, and fully ten percent of them are believed to be dangerous and leaking.

The result: not enough reliable environmentally safe places to dump toxic substances. Although the EPA wants to clean up as many landfills as possible, it has very little choice as to where to put the material it removes under the Superfund mandate. Taxpayers may wind up paying for the costly removal of waste from one site, only to find later on that they have to pay again for removing it from yet another dangerous site.

Meanwhile, legal fees mushroom. The litigation costs involving cleanup at the various Superfund sites are estimated to run somewhere between $3.5 billion and $6.4 billion.

Eventually, society will have to face the main reason for the scarcity of hazardous waste sites – the "not in my backyard" syndrome. Sites for the disposal of toxic substances have joined prisons and mental hospitals as things the public wants, but not too close by.

The hazardous-waste disposal problem is not going to disappear unless Americans adopt less polluting methods of production and consumption. Until then, greater understanding is needed on the part of the public, as is a willingness to come to grips with the difficult problems arising from the production and use of hazardous substances. It will cost large amounts of money (probably in the hundreds of billions of private and public expenditures in the next decade) to meet society's environmental expectations. Spending money may be the easiest part of the problem. Getting people to accept dump sites in their neighborhoods is much more difficult.

The answer surely is an appeal not merely to good citizenship, but also to common sense and self-interest. In a totalitarian society, people who do not want to do something the government desires are simply forced to do so, with the threat of physical violence ever present. In a free society with a market economy, we offer to pay people to do something they otherwise would not do. The clearest example in modern times is the successful elimination of the military draft coupled with very substantial increases in pay and fringe benefits for voluntarily serving in the armed forces.

Individual citizens have much to gain by opposing the location of hazardous waste facilities near them, and there is a basic logic to their position. It is not fair for society as a whole to benefit from a new disposal site, while imposing most of the costs (ranging from danger of leakage to depressed property values) on the people in the locality. But local resistance to dealing with hazardous wastes imposes large costs on society as a whole. Those costs are in the form both of inhibiting economic progress and having to ship waste from one temporary site to another.

Individual interests and community concerns can be reconciled by the use of economic incentives. The idea is to look upon environmental pollution not as a sinful act but as an activity costly to society and susceptible to reduction by means of proper incentives. After all, the prospect of jobs and income encourages many communities to offer tax holidays and other enticements to companies considering the location of a new factory – even though it may not exactly improve the physical environment of the region. Under present arrangements, however, there is no incentive for the citizens of an area to accept a

site for hazardous wastes in their vicinity, no matter how safe it is.

Some areas might accept such a facility if the state government (financed by all the citizens benefitting from the disposal facility) would pay for something the people in that locality want but cannot afford – such as a new school building, firehouse, or library, or simply lower property taxes. Unlike an industrial factory, a hazardous-waste facility provides few offsetting benefits to the local residents in the form of jobs or tax revenues. Government can do a lot to improve environmental policy in other ways. The EPA could reduce the entire hazardous waste problem by distinguishing between truly lethal wastes – which should be disposed of with great care – and wastes that contain only a trace or minute amounts of undesirable materials. To the extent that this would require changes in legislation, the agency should urge Congress to make them.

The Department of Defense now generates more than 500,000 tons of hazardous waste a year

The experience of a company in Oregon provides insights into why Congress needs to legislate common sense into the antipollution laws. The firm has been dumping heavy-metal sludges on its property for over 20 years. Company officials told the GAO that they automatically classify the material as hazardous. Why? Because it would be too costly and time-consuming to try to prove that it was not. The GAO learned from several industry associations that other companies, similarly uncertain and wanting to avoid expensive testing costs, simply declare their wastes to be hazardous, whether they really are dangerous or not. That is not the only example in which those complying with environmental regulations lose sight of the fundamental objectives to be met.

Tackling First Things First

A 1987 EPA report concluded that the agency's priorities "do not correspond well" with its rankings by risk of the various ecological problems on its agenda. The agency's own study found areas of high risk but little regulatory effort. A key example is runoff of polluted water from farms and city streets.

Conversely, the study showed that areas of "high EPA effort but relatively low risks" included management of hazardous wastes, cleanup of chemical waste dumps, regulation of underground storage tanks containing petroleum or other hazardous substances, and municipal solid waste. The reason for this mismatch between needs and resources is obvious. The EPA's priorities are set by Congress and reflect public pressure more than scientific knowledge. Driven by the forces of environmental politics, the nation has repeatedly committed itself to goals and programs that are unrealistic. This has meant deploying regulatory manpower unwisely and diverting limited resources to concerns of marginal importance.

The results of this mismatch are substantial. Not all hazards are created equal. Some disposal sites are being filled with innocuous material while truly dangerous substances are or will be, for lack of space, dumped illegally or stored "temporarily." What would help is more widespread application of the legal concept known as *de minimis non curant lex* – the law does not concern itself with trifles.

Back in 1979, a federal circuit court supported the view that there is a *de minimis* level of risk too small to affect human health adversely. It cited that doctrine in turning down the claim that some "migration" of substances occurred from the packaging into the food product. In 1985, the FDA concluded that using methylene chloride to extract caffeine from coffee presented a *de minimis* risk. The substance is safe for its intended use. In 1987, the National Research Council recommended that the EPA apply a "negligible risk" standard across the board in determining how much of which pesticides can be permitted to show up in food.

Cancerphobia Misallocates Resources

One approach to eliminating the gridlock in regulatory policy is to focus on the underlying public concern that is driving the pressures for more sweeping environmental and other social regulation. That concern is the worry about cancer. The regulatory waters have become badly muddied by the public's misconception of the causes of cancer. A widely held notion is that the environment is primarily responsible. There is, of course, a germ of truth to that belief.

It turns out that several years ago a distinguished scientist – John Higginson, director of the World Health Organization's International Agency for Re-

search on Cancer – assigned the primary blame for cancer to what he labeled "environmental" causes. His highly-publicized finding that two-thirds of all cancer was caused by environmental factors provided ammunition for every ecological group to push for tougher restrictions on all sorts of environmental pollution.

Upon a more careful reading, it is clear that the eminent scientist was referring not to the physical environment but to the age-old debate of "environment" versus "heredity" as the main influence on human beings. In the case of cancer, he was identifying voluntary behavior – such as personal life-styles and the kinds of food people eat – as the main culprit responsible for cancer. Higginson specifically pointed out, "But when I used the term environment i n those days, I was considering the total environment, cultural as well as chemical...air you breathe, the culture you live in, the agricultural habits of your community, the social cultural habits, the social pressures, the physical chemicals with which you come in contact, the diet, and so on." But that explanation has not slowed down the highly vocal ecology groups who latched on to a "catchy" albeit confused theme – the extremely carcinogenic environment in which Americans supposedly live.

More recently, one university scientist tried to add some objectivity to the cancer debate by quantifying the issue. Harry Demopoulos of the New York Medical Center examined why approximately 1,000 people die of cancer each day in the United States. About 450 of the deaths, or 45 percent, can be attributed to diet. Citing the work of Arthur Upton of the National Cancer Institute, Demopoulos noted that eating more fresh fruits and vegetables and curtailing fat consumption would be most helpful. Clearly, obesity is not the type of environmental pollution that justifies the EPA's increasingly onerous standards.

The second major cause of cancer deaths, according to Demopoulos, is the consumption of excessive quantities of distilled liquour and the smoking of high-tar cigarettes. These voluntary actions resulted in 350, or 35 percent, of the cancer deaths. Again, this is not the environmental pollution that motivates most ecology activists.

A distant third in the tabulation of leading causes of cancer is occupational hazards, accounting for five percent of the total. Demopoulos believes that this category may have leveled off and be on the way down. He reasons that many of the occupationally induced cancers are due to exposures two or more decades ago, when scientists did not know that many chemicals were capable of causing cancer.

A fourth category, accounting for three percent, is caused by exposure to normal background radiation. The fifth and last category of causes of cancer (accounting for two percent) is preexisting medical disorders. These include chronic ulcerative colitis, chronic gastritis, and the like. The remaining ten percent of the cancer deaths in the United States are due to all other causes; it is noteworthy that air and water pollution and all the toxic hazards that are the primary cause of public worry are in this miscellaneous ten percent, not in the 90 percent. Government policy is unbalanced when the great bulk of the effort deals with a category of risk that is only some fraction of one-tenth of the problem.

Hard data can dissipate much of the fear and fog generated by the many cancer-scare stories that the public has been subjected to in recent years. Overall, cancer death rates are staying steady or coming down. The major exception is smoking-related cancer. For the decade 1974-1983, stomach cancer was down 20 percent, cancer of the cervix-uterus was down 30 percent, and cancer of the ovary was down eight percent.

Life expectancy is steadily increasing in the United States (to an all-time high of 75, for those born in 1985) and in most other industrialized nations, except the Soviet Union. This has led cancer expert Bruce Ames of the University of California to conclude, "We are the healthiest we have been in human history." That is no justification for resting on laurels. Rather, Ames's point should merely help lower the decibel level of debates on environmental issues and enable analysis to dominate emotion in setting public policy in this vital area.

A Birth Control Approach to Pollution

Over 99 percent of environmental spending by government is devoted to controlling pollution after it is generated. Less than one percent is spent to reduce the generation of pollutants. For fiscal 1988, the EPA budgeted only $398,000 – or .03 percent of its funds – for "waste minimization." That is an umbrella term that includes recycling and waste reduction.

The most desirable approach is to reduce the generation of pollutants in the first place. Economists have an approach that is useful – providing incentives to manufacturers to change their production processes to reduce the amount of wastes

created or to recycle them in a safe and productive manner.

As we noted earlier, the government taxes producers rather than polluters. By doing that, the country misses a real opportunity to curb actual dumping of dangerous waste. The federal Superfund law is financed with taxes levied on producers of chemical "feedstocks" and petroleum plus a surtax on the profits of large manufacturing companies and contributions from the federal Treasury. Thousands of companies outside of the oil and chemical industries wind up paying very little, whether they are large polluters or not. Contrary to widely held views, a great deal of pollution occurs in sectors of the economy other than oil and chemicals. The manufacture of a single TV set generates about one hundred pounds of toxic wastes.

The pollution tax approach appeals to self-interest in order to achieve the public interest

Switching to a waste-end fee levied on the amount of hazardous wastes that a company actually generates and disposes of would be far more economically sound than the status quo. This more enlightened approach would require a basic correction in the Comprehensive Environmental Response, Compensation, and Liability Act (or "Superfund"), but it would be a very beneficial form of hazardous-waste "birth control."

A General Application of Market Incentives

More generally, if the government were to levy a fee on the amount of pollutants discharged, that would provide an incentive to reduce the actual generation of wastes. Some companies would find it cheaper to change their production processes than to pay the tax. Recycling and reuse systems would be encouraged. Moreover, such a tax or fee would cover imports which are now disposed of in our country tax-free. In short, rewriting statutes, such as the Superfund law, so that they are more fair would also help protect the environment – and would probably save money at the same time.

Already, some companies are recycling as they become aware of the economic benefits. One chemical firm burns 165,000 tons of coal a year at one of its textile fibers factories, generating 35,000 tons of waste in the form of fly ash. The company recently found a local cement block company that was testing fly ash as a replacement for limestone in making lightweight cement blocks. The chemical company now sells the fly ash to the cement block manufacturer. What used to be an undesirable waste by-product has been turned into a commercially useful material. The companies are simultaneously conserving the supply of limestone.

A timber company, through its research, developed a new use for tree bark, the last massive waste product of the wood products industry. The firm designed a bark processor that made it the first domestic producer of vegetable wax, an important ingredient in cosmetics and polishes. A factory in Illinois had been creating a veritable sea of calcium fluoride sludge (at a rate of 1,000 cubic yards a *month*) as a by-product of its manufacture of fluorine-based chemicals. The company found that the sludge could be mixed with another waste product to produce synthetic fluorspar, which it had been buying from other sources. Recycling the two waste products now saves the firm about $1 million a year.

Incentives to do more along these lines could be provided in several ways. The producers could be subsidized to follow the desired approach. In this period of large budget deficits, that would, of course, increase the amount of money that the Treasury must borrow.

A different alternative is to tax the generation and disposal of wastes. The object would not be to punish the polluters, but to get them to change their ways. If something becomes more expensive, business firms have a natural desire to use less of the item. In this case, the production of pollution would become more expensive. Every sensible firm would try to reduce the amount of pollution tax it pays by curbing its wastes. Adjusting to new taxes on pollution would be a matter not of patriotism, but of minimizing cost and maximizing profit. The pollution tax approach appeals to self-interest in order to achieve the public interest.

Charging polluters for the pollution they cause gives companies an incentive to find innovative ways to cut down on their discharges. These fees would raise costs and prices for products whose production generates a lot of pollution. It is wrong to view this as a way of shifting the burden to the public. The relevant factor is that consumer purchasing is not static. Consumer demand would shift to products

which pollute less – because they would cost less. To stay competitive, high-polluting producers would have to economize on pollution, just as they do in the case of other costs of production. Since pollution imposes burdens on the environment, it is only fair that the costs of cleaning up that pollution should be reflected in the price of a product whose production generates this burden.

The manufacture of a single TV set generates about one hundred pounds of toxic wastes

Nine countries in Western Europe have adopted the "polluter pays" principle. In these nations, pollution control is paid for directly by the polluting firm or from the money collected from effluent taxes. The West German effluent-fee system, the oldest in operation, began before World War I. It has succeeded in halting the decline in water quality throughout the Ruhr Valley, the center of West Germany's iron and steel production. It is also serving as a model for a more recent French effort.

Practical problems make changes in pollution policy difficult in the United States. Both the regulators and the regulated have an interest in maintaining the current approach. Pollution taxes have little appeal in the political system, particularly in Congress. Many reject a pollution tax on philosophical grounds, considering pollution charges a "license to pollute." They believe that putting a price on the act of polluting amounts to an attitude of moral indifference towards polluters. The tendency to look at ecological matters as moral issues makes it difficult to adopt a workable approach

Although economists are often accused of being patsies for the business community, environmental economics makes for strange alliances. So far, business interests have opposed the suggestions of economists for such sweeping changes in the basic structure of government regulation as using taxes on pollution. Despite the shortcomings of the present system of government regulation, many firms have paid the price of complying with existing rules. They have learned to adjust to regulatory requirements and to integrate existing regulatory procedures into their long-term planning.

As any serious student of business-government relations will quickly report, the debate over regulation is miscast when it is described as black-hatted business versus white-hatted public interest groups. Almost every regulatory action creates winners and losers in the business system and often among other interest groups. Clean air legislation, focussing on ensuring that new facilities fully meet standards, is invariably supported by existing firms that are "grandfathered" approval without having to conform to the same high standards as new firms. Regulation thus protects the "ins" from the "outs."

There are many other examples of regulatory bias against change and especially against new products, new processes, and new facilities. Tough emissions standards are set for new automobiles, but not for older ones. Testing and licensing procedures for new chemicals are more rigorous and thoroughly enforced than for existing substances. This ability to profit from the differential impacts of regulation helps to explain why business shows little enthusiasm for the use of economic incentives and prefers current regulatory techniques.

The reform of regulation is truly a consumer issue. The consumer receives the benefits from regulation and bears the burden of the costs of compliance in the form of higher prices and less product variety. The consumer has the key stake in improving the current regulatory morass.

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Exhibit K

Whereas, By Section 10 of an act of Congress, approved March 3, 1899, entitled "An act making appropriations for the construction, repair, and preservation of certain public works on rivers and harbors, and for other purposes," it is provided that it shall not be lawful to build or commence the building of any wharf, pier, dolphin, boom, weir, breakwater, bulkhead, jetty, or other structures in any port, roadstead, haven, harbor, canal, navigable river, or other water of the United States, outside established harbor lines, or where no harbor lines have been established, except on plans recommended by the Chief of Engineers and authorized by the Secretary of War; and it shall not be lawful to excavate or fill, or in any manner to alter or modify the course, location, condition, or capacity of, any port, roadstead, haven, harbor, canal, lake, harbor of refuge, or inclosure within the limits of any breakwater, or of the channel of any navigable water of the United States, unless the work has been recommended by the Chief of Engineers and authorized by the Secretary of War prior to beginning the same;

AND WHEREAS, By an instrument, dated May 12, 1898, the Secretary of War granted unto the Franklin Mining Company and the Arcadian Copper Company permission to dump sand from their stamp mills into Portage Lake (Torch Bay) at Grosse Point, Michigan, within certain limits, extending about six hundred feet from shore, as indicated on chart attached to said instrument, and subject to certain conditions therein set forth;

AND WHEREAS, The Franklin Mining Company and the Centennial Copper Mining Company, successor to the Arcadian Copper Company, have now applied to the Secretary of War for an extension of the limits, specified in said instrument of May 12, 1898, so as to permit the dumping of sand for a distance of about fifteen hundred feet from shore, within limits described as follows:

> Beginning at a point 1000 feet due south of triangulation station U, and running in a straight line to a point 1500 feet due south of station K, and thence by continuing said line and by a broken line following approximately the shore line at a distance therefrom of about 1500 feet to a point about 800 feet south of the buoy west of Grosse Point;

and as shown in red on the map hereto attached;

NOW, THEREFORE, This is to certify that, in accordance with the recommendation of the Chief of Engineers, the Secretary of War hereby gives permission, revocable at will by the Secretary of War, unto the said Franklin Mining Company and the Centennial Copper Mining Company, successor to the Arcadian Copper Company, to dump sand from their

stamp mills in Portage Lake at said place, within the limits shown on said map, and described above, subject to the following conditions:

1.---That the dump shall be built out as nearly vertical as possible so as to present a solid bluff to the bay.

2.---That the top of the embankment thus built shall not extend beyond the outer line.

3.---That this permission shall not be understood as authorizing any infringement of the rights of others, nor as conveying any riparian or other property rights not already vested in said grantees.

4.---That the work herein permitted to be done shall be subject to the supervision and approval of the Engineer Officer of the United States Army in charge of the locality.

WITNESS my hand this 10th day of JUNE, 1904.

Robert Shaw Oliver

Asst. Secretary of War.